HANDBOOK

OF

PRACTICAL MEDICINE

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VOLUME III

DISEASES OF THE NERVES, MUSCLES, AND SKIN

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HANDBOOK

OF

PRACTICAL MEDICINE.

SECTION V.

DISEASES OF THE NERVES.

PART I.

DISEASES OF THE PERIPHERAL NERVES.

A. DISEASES OF THE MOTOR NERVES.

a. PARALYSIS (AKINESIS) OF THE MOTOR NERVES.

1. *Paralysis of the Facial Nerve.*
(*Bell's Paralysis. Prosoplegia.*)

I. ETIOLOGY.—Peripheral facial paralysis is that form of disease whose causes are situated between the peripheral terminations of the nerve, and the point at which the trunk of the nerve enters the substance of the central nervous system, at the posterior edge of the peduncle of the pons. The paralysis also presents a peripheral character if the central fibres of the nerve are injured before they enter the nucleus in the floor of the fourth ventricle.

The peripheral course of the nerve may be divided into an intracranial portion, the part contained in the Fallopian canal, and the purely peripheral terminations.

Exposure occupies a prominent place among the causes of peripheral facial paralysis (rheumatic form). The paralysis sometimes follows the exposure at once, sometimes in a few hours or days.

The paralysis is often produced by riding in a car near an open window, looking out of a window when the face is warm, standing at a windy corner of a

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street, etc., and especially on that side which is exposed to the wind or cold air. Sleeping near a cold, damp wall is also sufficient to produce the paralysis.

In other cases traumatism is the cause of the paralysis.

This disease has been observed as the result of a slap in the face, the blow of a rapier, the application of the forceps during delivery. Tumors of the pelvis, and a narrow pelvis are also said to produce facial paralysis in the new-born. Blows, stab-wounds, gunshot wounds, may paralyze the nerve. The disease also results not infrequently from surgical operations upon the parotid gland, and it has been observed after stretching of the nerve in convulsive tic. It is sometimes the result of severe injury to the skull, with subsequent fracture and hemorrhage, since an accumulation of blood in the Fallopian canal is apt to impair the function of the nerve.

Diseases of the parotid gland, enlargement of the submaxillary lymphatic glands, abscesses in the region of the lower jaw, or suppuration of tubercular lymphatic glands, with subsequent formation of cicatrices, may also give rise to the paralysis, either as the result of compression or direct implication of the nerve in the morbid process. May described facial paralysis in leukæmia from leukæmic infiltration of the nerve.

The disease is often the result of aural affections.

According to Graig, the paralysis may be caused by an accumulation of wax in the ear, and may rapidly disappear when the cerumen is removed. It has also been assumed that catarrhal inflammation of the cavity of the tympanum may implicate the facial nerve. The connection between tubercular processes in the temporal bone and facial paralysis is clearer, because the destructive process is apt to extend to the trunk of the nerve. The paralysis is sometimes produced by neoplasms or hemorrhages in the Fallopian canal.

In certain cases intracranial diseases are the causes of peripheral facial paralysis, for example, meningitic changes, exostoses, tumors, aneurisms of the vessels at the base of the brain, etc.

The infectious diseases occasionally act as a cause (diphtheria, erysipelas, typhoid fever, variola, and dysentery). Several cases have also been described after herpes zoster. Peripheral facial paralysis is not infrequently the result of syphilis. The lesion is sometimes intracranial (chronic meningitic processes, exostoses, or gummata), sometimes it consists of an exudative process within the Fallopian canal. French writers assert that the paralysis sometimes occurs temporarily at the beginning of the secondary stage of syphilis, particularly in females.

The disease is said to have been observed in lead poisoning, and also after over-exertion of certain facial muscles.

The disease is an unusually frequent one. It is observed more often in men because they are more exposed to the various causes, and is more frequent in middle life for a similar reason. Eulenburg observed a patient who had had the disease twice on the right side, and three times on the left side.

II. ANATOMICAL CHANGES.—I have had the opportunity of making a post-mortem examination of a case of facial paralysis, following tubercular disease of the petrous portion of the temporal bone. The changes were identical with those observed shortly after section of a nerve in animals: degeneration of the medullary substance into small fragments,

destruction of the axis cylinders, increase of the nuclei in the sheath of Schwann, increase of the interstitial connective tissue and of its nuclei (Fig. 1). Similar appearances were described by Kaase in a case of six weeks' standing. In this case the frontal muscle showed fatty degeneration of the fibrillæ, increase of the interstitial connective tissue, and an accumulation of fat within it.

These changes must always be expected when the lesions which have given rise to the facial paralysis have interrupted the continuity of the nerve fibres. If the causes disappear, the anatomical changes may be repaired. The restoration of the parts may remain absent if the lesion has lasted too long or if a large part of the nerve has been entirely destroyed.

Fig. 1.

Degenerated nerve fibres from the facial nerve, which was paralyzed as the result of tuberculosis of the petrous portion of the temporal bone. Osmic acid preparation. Enlarged 275 times.

In mild, temporary paralyses, the lesions are probably less severe. In not a few cases, they consist probably of inflammatory swelling and changes in the connective-tissue portions of the nerve, which temporarily compress the nerve fibres and impair conduction. As a matter of course, such conditions will be more serious if situated within the Fallopian canal, on account of its narrowness and unyielding character.

III. SYMPTOMS.—The symptoms of facial paralysis are not confined to purely motor disturbances in all cases. According to some authors, the facial nerve contains secretory fibres at its origin from the pons, so that it is not surprising that disturbances in the secretion of saliva may occur in facial paralysis. At the ganglion geniculatum, the facial nerve receives gustatory nerve fibres from the trigeminus through the agency of the petrosus superficialis major (but the gustatory fibres soon

leave the facial nerve with the chorda tympani), and hence the symptoms of facial paralysis may include disturbances of taste.   Finally, since the facial nerve innervates the stapedius muscle through the agency of the stapedius nerve, auditory disturbances may also be produced.

The symptoms of facial paralysis may occur suddenly or be preceded by prodromata (pain in the ear and face, dizziness, etc.).   In some cases, long-standing otorrhœa and the signs of tuberculosis of the petrous portion of the temporal bone prepare us for the possible onset of facial paralysis.

The first symptoms are often noticed by the patient's family or accidentally by himself on looking into a mirror.   Sometimes the patient complains merely of imperfect closure of the lid and epiphora, although the paralysis must have been present for several days.   Some patients

FIG. 2.
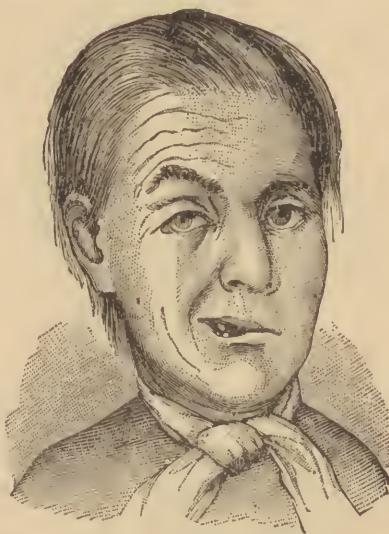
Expression of the face, during the act of laughing, in a woman suffering from peripheral paralysis of the left facial nerve.

complain of a feeling of weight, stiffness, or coldness on one side of the face, or of a sour, salty, or metallic taste upon one-half of the tongue.

The motor disturbances are the most prominent symptoms.   The affected half of the face is destitute of folds, smooth, drawn towards the healthy side, and remains motionless during laughter, crying, or talking (Fig. 2).

On account of paralysis of the frontal muscle, the forehead is somewhat higher on the affected than on the healthy side; it is smooth, and remains so when the attempt is made to wrinkle it.

On account of paralysis of the corrugator supercilii, the glabella on the affected side remains smooth when an attempt is made to wrinkle the integument in this region.   The eyebrow is lower than on the healthy side.

Paralysis of the orbicularis palpebrarum causes the upper lid to

appear smaller than on the healthy side, and hence the palpebral fissure to appear larger. The eyeball is sometimes more prominent. The patient is unable to close the lid (lagophthalmus paralyticus), though the palpebral fissure becomes somewhat narrower in the attempt.

Inasmuch as the eyeball is rolled upwards and inwards (more rarely upwards and outwards) by associated movements, the sclera and the lowermost segment of the iris make their appearance upon attempting to close the paralyzed lid. All these movements are usually spasmodic and irregular.

The explanations of the diminution in the size of the palpebral fissure during the attempt to close the lid are unsatisfactory. Some assume an unequal paralysis of the various layers of the orbicularis. Henle believes that the levator palpebræ relaxes, contrary to the rule, during the attempt at closure, and thus renders possible the phenomenon referred to.

The patients are very much annoyed by constant epiphora, the result of paralysis of Horner's muscle, which changes the position of the lachrymal points in such a way that the lachrymal fluid cannot enter them freely. After a while, the paralysis may be associated with conjunctivitis, ulceration of the cornea, or even deeper-seated diseases of the eye. Marked ectropium paralyticum of the lower lid develops not infrequently.

The tip of the nose is directed towards the healthy side. The nostril on the affected side is smaller than that on the other side on account of the paralysis of the muscles which move the ala nasi. The patient often complains of a peculiar feeling of dryness in the affected side of the nose and diminished sense of smell, the result of dryness of the mucous membrane on account of the imperfect flow of tears through the lachrymal canal, and of the diminished size of the nostril.

The naso-labial fold is lessened or abolished on the paralyzed side. The angle of the mouth is depressed and drawn towards the healthy side; this is also true of the chin. The mouth is more or less open on the paralyzed side, and often permits the escape of saliva and fluids. For this reason, many patients, in drinking, throw the head backwards and towards the healthy side. Articulation is interfered with on account of the imperfect closure of the lips. This is most marked in the enunciation of the labials, the paralyzed cheek flapping to and fro in the attempt. Whistling, blowing, expectoration are also interfered with or rendered impossible, because the air escapes from the paralyzed side of the mouth.

The movements of the tongue and of deglutition are undisturbed (the stylohoid and biventer mandibulæ muscles are supplied by the facial nerve, but are of such minor importance that their paralysis is unnoticed).

The apparent deviation of the tongue when protruded is simply the result of the drawing of the mouth towards the healthy side.

Although the movements of mastication are unaffected, this function is interfered with to a certain extent by the paralysis of the buccinator muscle. Food may accumulate between the gums and the cheek, and must then be removed with the fingers or by pressure upon the cheek from the outside.

Paralysis of the branches which supply the muscles of the concha is

recognizable, on inspection, in those individuals who possess the power of moving the ear, or by electrical examination of the muscles.

The sensibility of the integument is almost always intact. with the exception of the rare cases in which the cause of paralysis is situated so far peripherally that the inosculating peripheral ramifications of the trigeminus are also affected.

Vaso-motor disturbances are never observed. Berger reports one case in which the hair turned gray upon the paralyzed side.

Reflex and associated movements are always absent in peripheral as opposed to central facial paralysis.

Auditory disturbances are observed under certain circumstances. If a peripheral facial paralysis is the result of intracranial processes or tubercular and inflammatory changes in the petrous portion of the temporal bone, audition may be impaired or abolished, either because the acoustic nerve has also been injured or on account of the destruction of the auditory apparatus within the tympanic cavity. On the other hand, the facial paralysis in itself may be associated with increased power of hearing (hyperacusis, oxyokoia). Hitzig also mentions that some patients hear a low note upon voluntary contraction of the frontal muscle. These phenomena are explained by the fact that the stapedius muscle is supplied by the facial nerve. If this muscle is paralyzed, the tensor tympani assumes the upper hand and produces the hyperacusis (the modus operandi has not been satisfactorily explained).

Disturbances of taste are possible, owing to the fact that the facial nerve receives gustatory fibres, at the ganglion geniculatum, through the nervus petrosus superficialis major. These fibres leave the facial nerve in the chorda tympani. so that disturbance of taste will occur only when the lesion is situated between the ganglion geniculatum and the exit of the chorda tympani. The patients complain not infrequently of subjective perverse sensations of taste (salty, metallic, etc.). In severe cases the patients lose the sense of taste entirely (ageusis). or it is distinctly diminished or perverted in the anterior two-thirds of the tongue on the paralyzed side (the posterior third is supplied by the glosso-pharyngeal nerve).

In testing the sense of taste, the tongue should be protruded (the eyes being closed), and its tip and edge brushed with various salty, sweet, sour, and bitter solutions. The tongue should not be withdrawn into the mouth during the experiment. Taste may also be tested with the galvanic current. A wire electrode, terminating in a small button, should be employed (vide Vol. I., Fig. 30). The object of the examination is to determine whether the first sensation of taste is produced on both sides by the same number of elements on touching the tongue with the electrode, and whether the patient is able to discern that the anode produces a more intense gustatory sensation of a more metallic, sour taste. while the action of the cathode is less intense, and the gustatory sensation more biting and salty. The indifferent electrode, *i. e.,* the one which is not in contact with the tongue, may be placed upon any indifferent spot, for example. the sternum. The test is begun with one element, and the strength of the current gradually increased, if necessary. The prickling sensation produced by the current must be distinguished from gustation.

The tactile sensibility of the tongue is not infrequently diminished upon the paralyzed side.

Many patients complain of diminished salivary secretion and a feeling of dryness in the affected half of the buccal cavity.

The connection of the ganglion geniculatum with the spheno-palatine ganglion, through the agency of the nervus petrosus superficialis major, also explains the possible occurrence of paralysis and changes in position of the uvula and soft palate. The motor fibres in question pass from the facial into the palatine nerve. On opening the mouth, the arch of the palate is found depressed on the paralyzed side, and flutters to and fro on forced respiration. The tip of the uvula almost always projects towards the healthy side, but it must be remembered that deflections of the uvula may occur in the healthy subject.

The electrical reactions of the paralyzed muscles are very important with regard to diagnosis and prognosis. Facial paralysis may be divided into mild, moderately severe, and severe forms, according to the character of the electrical reactions.

In the mild form, there are hardly any changes in the electrical irritability to the faradic and galvanic currents, the muscles reacting (both directly and indirectly) as well as on the healthy side. In some cases, indeed, th indirect electrical irritability is increased for a few days after the paralysis, so that a milder current suffices to produce minimal contractions of the paralyzed muscles. Paralyses which present such reactions until the seventh day offer a very favorable diagnosis. They usually recover within two or three weeks without special treatment.

The moderately severe form of facial paralysis presents the partial degeneration reaction. If electricity is applied to the nerve (indirect stimulation), a slight increase of excitability may be noticeable for a few days after the paralysis, but towards the end of the first week the faradic and galvanic excitability diminishes more and more, as is shown by the fact that the muscular contractions on the paralyzed side are less vigorous than on the healthy side, although the minimal contractions often are produced by the same current on the two sides. The electrical reactions on direct stimulation of the muscles are entirely different. They present qualitative and quantitative changes which grow more profound during the second and third weeks of the disease. Towards the end of the first week the faradic reaction gradually diminishes. The galvanic excitability also diminishes somewhat during the first week, but it grows more active during the second week. The mildest current is often sufficient to produce muscular contractions on the paralyzed side. But these contractions are slow, prolonged, and feeble, and are apt to become tetanic, often during the entire duration of the passage of the current. At the same time the law of normal contraction changes. If a healthy muscle is stimulated with an increasing strength of current, the contractions will occur in the following order:

Cathodal closure contraction (CaClC).
Anodal closure contraction (AnClC).
Anodal opening contraction (AnOC).
Cathodal opening contraction (CaOC).

In peripheral paralysis, AnClC follows CaClC very rapidly at the beginning, soon it appears with the same strength of current, and finally AnClC exceeds CaClC. This is also true of CaOC which soon becomes equal to AnOC, though it rarely exceeds the latter.

The moderately severe form usually recovers in four to six weeks, at the latest in eight to ten weeks. It disappears without leaving any

sequelæ (twitchings or contractures). Voluntary power often returns before the changes in the galvanic excitability have disappeared.

In the severe form of the disease, complete degeneration reaction is observed. The excitability of the nerve to both currents diminishes at once or after a slight increase of excitability for one or two days. This diminution of excitability spreads from the central portions of the nerve to the periphery, and finally the electrical irritability disappears entirely. It may remain absent for weeks and months or, in incurable cases, permanently. Direct stimulation of the muscles, on the other hand, reveals the changes which we mentioned above in our description of the moderately severe form.

FIG. 3.
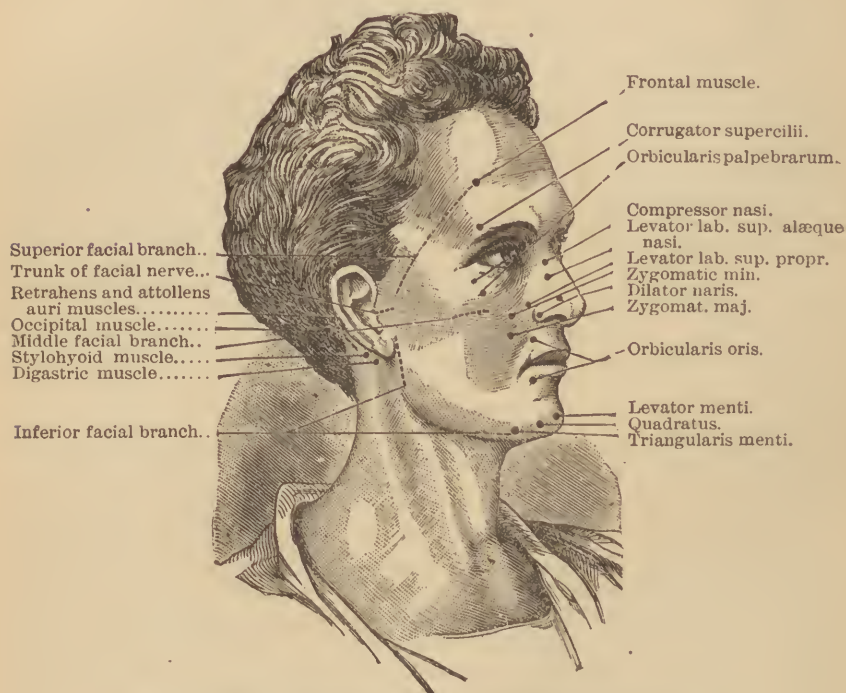
Motor points of the facial nerve and the facial muscles supplied by it.

In addition to an increase of galvanic excitability, the muscles also manifest increased mechanical excitability.

In many cases of severe facial paralysis, a period of one to one and one-half years elapses before recovery occurs. At all events, voluntary contractions are hardly ever observed before the end of the second or third month. Recovery is not infrequently incomplete; twitchings and contractures persist in the previously paralyzed muscles. Voluntary power returns before the indirect electrical excitability, i. e., the nerve fibres have become capable of conducting the stimulus of the will before they react to peripheral stimuli. In the incurable cases, the increased excitability to the galvanic current gradually disappears, and the muscles finally become altogether inexcitable.

The changes of electrical excitability which we have described depend upon definite anatomical changes in the nerves and muscles. Experiments on animals prove that when a peripheral nerve is cut, the medulla and axis cylinder of the fibres in the peripheral portion undergo degeneration and become incapable of conduction. The more the degeneration of the nerve progresses, the more its electrical excitability diminishes. Anatomical changes also appear in the muscles supplied by the cut nerve. About the second week, the muscular fibres become narrower, the transverse striation less distinct, the muscle nuclei increase in number, and the interstitial connective tissue proliferates. E. Neumann showed that muscles in this condition lose the power to react to currents of short duration (faradism), but react to galvanic currents of sufficient duration in the manner known as degeneration reaction.

After the degeneration of the nerve has lasted for some time, a regeneration of the fibres occurs, if the conditions are favorable. At first, the new fibres are destitute of medulla, and are unable to conduct peripheral impulses to the mus-

FIG. 4.

Bilateral facial paralysis. Face in profile. After Wright.

cles, although they conduct the will power. The electrical excitability appears to be dependent on the presence of the medulla.

In the mild form, there is probably no anatomical change in the nerve or muscle; in the moderately severe form the anatomical disturbances in the nerve are only temporary, but the muscular tissue presents more marked lesions. In incurable cases the atrophy of the muscular fibres and increase of the connective tissue become so excessive that the electrical excitability is entirely lost.

Motor points are those places from which a muscle can be made to react most readily to electrical stimulation. The motor points of the facial muscles are shown in Fig. 3.

As a rule, peripheral facial paralysis is unilateral, and affects almost all branches of the nerve.

Bilateral facial paralysis (diplegia facialis) is rare. It is sometimes

found that, after exposure, at first one nerve is paralyzed, and after a few days the other facial nerve is also paralyzed. Or bilateral paralysis may be the result of lesions of the petrous portion of both temporal bones, or of intracranial processes. Maingauld described diplegia facialis after diphtheria. Sometimes the paralysis on one side is peripheral, on the other side it is central in its origin.

Persons suffering from diplegia facialis have entirely lost the power of mimetic movements. We may hear the patients laugh or cry, but their features remain rigid and set. There is inability to close the lids, the mouth is open, the upper lip often hangs down like a snout, saliva dribbles from the mouth. Speech is indistinct and nasal. Deglutition is impaired, and many patients are compelled to push the food, with the fingers, back of the palate.

Fig. 5.

Facial expression in bilateral facial paralysis. Full face. After Wright.

Various causes may give rise to incurability of the paralysis, viz., tubercular processes in the petrous portion of the temporal bone, operations, injuries, etc. In certain cases, recovery is incomplete. The muscles remain paretic, and twitchings and contractures are observed. If the paralysis is permanent, the facial muscles and integument undergo atrophy.

IV. DIAGNOSIS.—The recognition of facial paralysis as such is not difficult, and, as a rule, it is also easy to differentiate the central from the peripheral form.

It is characteristic of central facial paralysis that:

a. The movements of the forehead and eyelid are usually unaffected, except when the disease is the result of a lesion of the pons which affects the course of the facial nerve on the peripheral side of the facial nucleus.

b. The electrical excitability is unchanged or is even increased for a few days.

c. There is usually paralysis of the limbs on the same side, except in certain cases of pons lesion, in which the limbs are paralyzed on the opposite side of the body.

d. According to Strauss, the secretion of perspiration after subcutaneous injections of pilocarpin is unaffected on the paralyzed side in central facial paralysis, but is delayed and diminished in peripheral facial paralysis.

The following anatomical details must be considered with regard to the situation of the paralysis (vide Fig. 6):

1. In paralysis whose causes are outside of the stylo-mastoid foramen, the symptoms will consist simply of paralysis of the facial muscles (vide Fig. 6, 1).

FIG. 6.
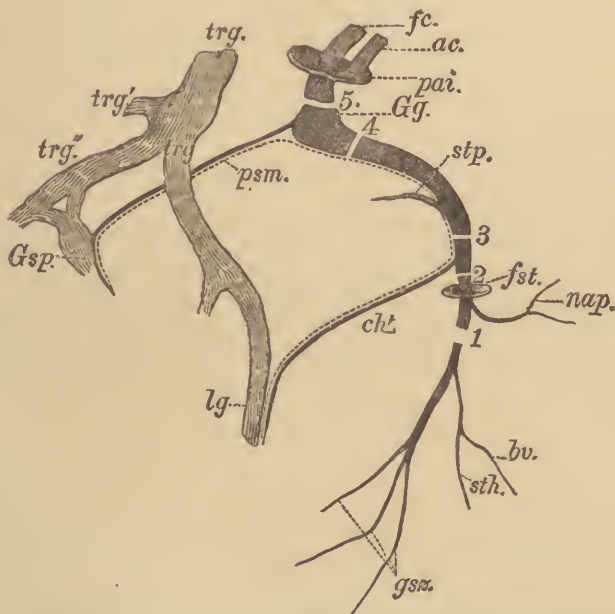
Schematic representation of the course of the facial nerve.

fc, trunk of facial nerve; *ac,* trunk of acoustic nerve; *pai,* internal auditory foramen; *Gg,* geniculate ganglion; *psm,* nervus petrosus superficialis major; *stp,* stapedius nerve; *cht,* chorda tympani; *fst,* styloid foramen; *nap,* post. auricular nerve; *bv* and *sth,* nerves to the digastric and stylohyoid muscles; *trg, trg', trg'', trg''',* trunk and the three branches of the trigeminus; *Gsp,* sphenopalatine ganglion; *lg,* lingual nerve.

2. If the cause of paralysis is situated in the lower part of the Fallopian canal, but below the point of departure of the chorda tympani, the paralysis of the facial muscles is associated with paralysis of the muscles of the ear and the occipitalis muscle, which are supplied by the posterior auricular nerve (vide Fig. 6, 2).

3. If the lesion is situated within the canal between the origin of the stapedius and chorda tympani nerves, we will find: paralysis of the facial muscles, the muscles of the ear and occipitalis, disturbances of taste and salivary secretion (vide Fig. 6, 3).

4. If the lesion is situated between the ganglion geniculatum and

the origin of the stapedius nerve, the symptoms produced are: paralysis of the muscles of the face, external ear, and the occipitalis, disturbances of taste, salivary secretion, and audition (vide Fig. 6, 4).

5. If the lesion is situated immediately above the nervus petrosus superficialis major, or at the ganglion geniculatum itself, paralysis of the palate will be associated with the symptoms mentioned in the preceding section.

6. If the lesion is situated above the ganglion geniculatum and in the intracranial portion of the nerve, disturbances of taste will be absent, but paralysis of the facial muscles, occipitalis, external ear muscles, disturbances of hearing and salivary secretion, and palatal paralysis will remain. In this form of disease, the adjacent nerves are often paralyzed, for example, the abducens, and particularly the acoustic.

V. Prognosis.—The prognosis depends in the first place upon the cause. If the latter cannot be relieved, there is no hope of recovery of the paralysis. Paralyses of the facial nerve which occur during delivery recover spontaneously in one to two weeks, and rarely interfere with nursing.

As a general thing, the prognosis is more favorable in young persons, Above all, however, it depends on the condition of the electrical excitability (vide page 7).

VI. Treatment.—Causal treatment should first be instituted. For example, syphilitic lesions must be treated with mercury and iodine. Compressing tumors and abscesses must be treated according to surgical principles. In rheumatic facial paralysis we may recommend hot baths and iodide of potassium (℥ iij. : ℥ vij., one tablespoonful t. i. d.).

Local treatment must also be adopted. Not much can be expected from applications of iodine, leeches, etc. Good results are said to have been obtained from subcutaneous injections of strychnine in certain forms of paralysis, especially after diphtheria. But real benefit is obtained from electrical treatment alone. However, our hopes must not be pitched too high, since the anatomical changes in the nerve and muscles will run their course despite all treatment. It appears possible to shorten the duration of the disease only in so far as the electrical treatment of the nerves may hasten the removal of the products of inflammation, and the direct stimulation of the muscles counteracts the atrophic changes.

If the paralysis is situated in the Fallopian canal or the intracranial portion of the facial nerve, the galvanic current should be passed transversely through the head, the anode being placed immediately in front of the mastoid process on the affected side, the cathode on the corresponding part on the opposite side. Each sitting should last two to five minutes, and be repeated three to four times a week. The current should not be strong enough to produce pain or vertigo.

If the paralyzed muscles react to the faradic current, this should be applied to each individual muscle, so that the latter undergoes several successive contractions. The indifferent pole is applied to the sternum, the other to the motor points of the muscles (vide Fig. 3). The electrode applied to the motor points should be small, thoroughly moistened, and firmly applied. Strong currents must be avoided, since they are apt to give rise to electrical contracture of the muscles and serious deformity of the face. The duration of the entire sitting should not be longer than three or five minutes. Some physicians recommend the application of the faradic current even if the paralyzed muscles have lost their faradic excitability. In such cases we prefer the galvanic current. The indif-

ferent pole (preferably the anode) is applied to the sternum, the cathode is drawn across the individual muscles (so-called labile application). If the faradic irritability of the muscles returns as the paralysis improves, we may use the faradic and galvanic currents alternately.

In old cases of peripheral facial paralysis, Rosenthal recommended intra-buccal galvanization, i. e., the anode is placed on the mucous membrane of the cheek, the cathode externally upon each individual muscle. Landois and Mosler employed the simultaneous application of the galvanic current to the nerve and the faradic current to the muscles.

If contractures and spasmodic twitchings persist in the muscles after peripheral facial paralysis, we may endeavor to relieve the contracture by the application of stabile constant currents (one pole on the mastoid process, the other fixedly on the pes anserinus or the individual muscles). Stretching or massage of the contractured muscles may also be recommended. Many patients attempt to relieve themselves by introducing small globes between the cheek and jaw. Myotomy and tenotomy of the shortened muscles have been occasionally resorted to, or the attempt has been made to compensate for the facial deformity by producing vigorous faradic irritation and electrical contracture of the healthy muscles.

2. Motor Trigeminus Paralysis.

(Paralysis of the Muscles of Mastication.)

1. The third branch of the trigeminus contains only motor fibres. These form a separate nerve root at the point at which the trigeminus emerges from the brain near the anterior border of the middle peduncle; this root is smaller than the sensory trigeminus root, and is usually situated immediately in front of the latter. The inframaxillary branch leaves the skull through the foramen ovale to pass to the muscles of mastication—the temporal, masseter, external and internal pterygoids, mylo-hyoid, anterior belly of the digastricus, tensor tympani, and spheno-staphylinus.

Peripheral paralyses in the distribution of the third branch of the trigeminus are exceedingly rare. During its extracranial course the nerve is situated so deep that it is with difficulty accessible to refrigeratory (rheumatic) influences. Furthermore, tubercular, syphilitic, or other diseases of the sphenoid bone are so uncommon that they are only exceptional causes of paralysis. The most frequent causes of peripheral paralysis of this nerve are intracranial diseases—meningitis, syphilitic changes, tumors, and aneurisms. At the same time, the first and second branches of the nerve are often implicated, and the disease often extends to adjacent cerebral nerves—for example, the facial or abducens.

2. In the disease under consideration there is loss of power in the muscles of mastication. During this act, the projection of the temporal and masseter regions remains absent, and upon applying the fingers we miss the hard swelling which is usually felt over the contracting muscles. On account of paralysis of the pterygoids, the jaw is deflected toward the paralyzed side during each movement of mastication. The patients are unable to move the jaw voluntarily towards the healthy side. They attempt by the aid of the tongue to push the bolus of food between the jaws on the unaffected side.

Changes in the position of the palate and auditory disturbances on

account of the paralysis of the spheno-staphylinus and tensor tympani muscles are conceivable, but have not been recognized with certainty. According to Lucae, paralysis of the tensor tympani is shown by the fact that the patient hears a low murmur subjectively, but is unable to recognize low notes objectively.

There are usually disturbances of sensation. These may be confined to the third branch, or affect all branches of the trigeminus (vide article on Anæsthesia of the Trigeminus).

The electrical reactions of the paralyzed muscles correspond to those observed in ordinary peripheral paralysis. Little is known concerning the reaction of the paralyzed nerve, on account of its deep situation.

Atrophy and contracture of the muscles may develop after paralysis of long standing. The lower jaw is then drawn strongly upwards.

Fig. 7.
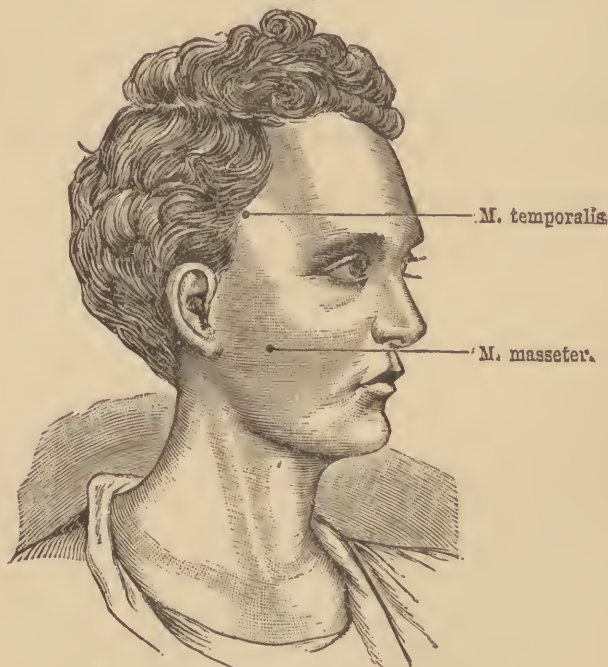
Motor points of the masseter and temporal muscles.

if the paralysis is bilateral, the lower jaw drops and is immovable. This is often noticed during the final agony.

3. The treatment must be etiological and local. The former depends upon the causation, the latter consists of electrical applications. If the disease is the result of intracranial lesions, the constant current should be passed transversely through the skull. In addition, applications should be made directly to the paralyzed muscles. The motor points are shown in Fig. 7.

3. *Paralysis of the Spinal Accessory Nerve.*

1. The spinal accessory nerve is composed of numerous nerve roots, of which the lowermost may be followed into the spinal cord to the

level of the sixth and seventh cervical vertebræ, while the uppermost emerge from the medulla oblongata. After the roots have united into the trunk of the nerve, they leave the skull through the jugular foramen. The nerve then divides into an anterior, internal branch, and a posterior, external branch. The former joins the pneumogastric, and passes into the recurrent laryngeal, pharyngeal, and cardiac branches of the vagus. The external branch supplies motor fibres to the sternomastoid and trapezius. These muscles also receive motor fibres from the cervical plexus. According to Bernard, the external branch of the spinal accessory is composed chiefly of fibres of spinal origin.

The symptoms of paralysis of the nerve are most distinct when the

FIG. 8.

Motor points of the sternomastoid and trapezius muscles and the spinal accessory nerve.

external branch is alone affected; that of the internal branch will not be considered here.

The causes of peripheral paralysis of the external branch include: cold, wounds, tumors, and abscesses in the neck, diseases of the cervical vertebræ, rarely intracranial lesions.

2. In unilateral paralysis of the sternomastoid muscle, the head is held somewhat obliquely (caput obstipum paralyticum). The face looks towards the paralyzed side; the chin is turned towards the same side, and is directed slightly upwards (unopposed action of the healthy muscle). Passive motion of the head from before backwards is possible, but the active motion is performed only with the aid of auxiliary muscles.

If the paralysis has lasted a long time, the muscle undergoes in-

creasing atrophy. The healthy sternomastoid undergoes permanent contraction, and caput obstipum spasticum gradually develops.

When both muscles are paralyzed, the head is held straight, but its motion backwards and upwards is rendered very difficult, and the projection of the muscle on each side of the neck remains absent.

In unilateral paralysis of the trapezius, the scapula is lower on the paralyzed than on the healthy side. It is also farther removed from the spine, the upper inner angle being farther removed than the lower angle. The weight of the arm draws the upper outer angle of the scapula forwards and downwards, and this effect is increased still more by the contraction of the levator anguli scapulæ and rhomboid muscles. The supraclavicular fossa is unusually deep. The raising of the shoulder is interfered with, and also the approximation of the scapula to the spine. Elevation of the arm above the horizontal is also interfered with, because the scapula is imperfectly fixed upon the thorax.

In bilateral paralysis of the trapezii, the back appears unusually broad and curved. The head is usually inclined towards the chest.

In combined paralysis of the sternomastoid and trapezius, the symptoms include all those described above.

If the trunk of the nerve is paralyzed, the symptoms of paralysis of the palate and of the recurrent laryngeal nerve are superadded (vide Vol. I., page 196). Increased frequency of the pulse has been observed in bilateral paralysis of the nerve (the cardiac branches of the vagus are derived in part from the spinal accessory).

The electrical reactions of the paralyzed nerve and muscles correspond to those described with regard to facial paralysis.

3. Treatment includes the removal of the cause, and the application of electricity. If the disease is the result of intracranial causes, the galvanic current should be passed transversely through the skull; in addition, electricity should be applied to the paralyzed muscles. The motor points and the superficial course of the nerve are shown in Fig. 8. Orthopædic measures and myotomy are indicated in old cases, associated with contracture.

4. Paralysis of the Hypoglossal Nerve.

(Glossoplegia.)

1. The hypoglossal nerve emerges from the groove between the olivary body and pyramid—the prolongation of the anterior lateral furrow of the spinal cord. After the roots have united into a trunk, the latter passes through the anterior condyloid foramen, and is distributed to the tongue muscles (hypoglossus, genioglossus, styloglossus, lingual), to the geniohyoid, omohyoid and sternohyoid, and to the sternothyroid and hyothyroid muscles.

In unilateral paralysis of the hypoglossus, the tongue, when protruded, deviates to the paralyzed side, on account of the unopposed action of the normal genioglossus muscle. The paralyzed half of the tongue is more deeply furrowed, and often presents lively fibrillary contractions ; it often undergoes atrophy, after the paralysis has lasted for some time.

The disturbances in the movements of the tongue are especially noticeable in mastication and talking. The formation of the bolus is imperfect, and particles of food often remain upon the paralyzed side. On account of the diminished mobility of the bolus in the mouth, many

patients complain of diminished gustatory sensation.   The tongue normally separates the buccal from the pharyngeal cavity during deglutition.   This is imperfectly effected in paralysis of the tongue, so that food and drink regurgitate into the buccal cavity during deglutition. For the same reason the patients experience difficulty in swallowing saliva, and often expectorate it.

In some cases the chief symptom is difficulty in the articulation of linguals, *l. s, sch*, and also of *k, g, ch, r. u, s, f.*   Singing (the production of high and falsetto notes) is also interfered with, because these require associated movements of the tongue.

In bilateral hypoglossal paralysis, the tongue is motionless in the buccal cavity, and the disturbances of speech and mastication become

FIG. 9.

Motor points of the hypoglossus nerve and the muscles supplied by it.

more marked.   The saliva sometimes flows almost uninterruptedly from the mouth.

The electrical reactions are the same as in other peripheral paralyses.

2. Peripheral paralysis of the hypoglossus is rare.   Gendrin reported a case in which the nerve was compressed by hydatids within the condyloid foramen.   Weir Mitchell observed it after a gunshot wound.   It has also been observed as the result of operations or the growth of tumors.

3. Treatment consists in the main of the application of electricity. The motor points of the muscles are shown in Fig. 9; the peripheral trunk of the nerve is found immediately above and behind the greater cornu of the hyoid bone.   In addition, electricity may be applied di-

2

rectly to the tongue. The most suitable electrode for this purpose is the one depicted in Vol. I., Fig. 50. In galvanization of the trunk of the nerve, the cathode is applied to the point shown in Fig. 9, the anode to the back of the neck.

5. *Paralysis of the Radical Nerve.*

I. ETIOLOGY.—Among the peripheral paralyses of the nerves of the arm, that of the radial nerve occurs most frequently. Its long and superficial course makes it easily accessible to rheumatic and traumatic influences. The most frequent cause is traumatism; cold (rheumatic or refrigeratory paralysis) is a rarer etiological factor. Isolated radial paralysis is observed not infrequently in hysteria and lead poisoning, but it is not probable that this is the result of peripheral disturbances.

The disease also occurs after infectious diseases (typhus fever, acute articular rheumatism). In rare cases it seems to be the result of overexertion of the muscles supplied by the nerve.

The most frequent form of peripheral radial paralysis is the so-called " sleep " paralysis. It develops in individuals who, while drunk or worn out, have fallen asleep in a position in which they pressed upon the outer surface of the arm and with it the radial nerve, especially at the bend in its course. The patients sometimes fell asleep with the head resting on the arm, and the latter pressing upon the sharp edge of the bed, etc.; sometimes the arm was drawn alongside the chest, and the protracted lateral decubitus and weight of the body produced the compression; or the arm was drawn up under the head. The paralysis is more frequent on the right side, because most people are accustomed to sleep upon the right side of the body. The more profound the sleep, the greater the danger of paralysis. It rarely occurs after natural sleep at night, but usually in drunkards or in laborers who take a short nap at noon. The paralysis is usually present on awaking, in rarer cases the patients first complain of numbness and tingling in the forearm, and this is followed in a few hours by paralysis. The triceps and the cutaneous sensibility of the arm and forearm are unaffected, because the compression generally takes place below the point at which the nerves to these parts are given off. If the pressure has been produced upon the upper third of the dorsal surface of the forearm, the supinator longus will not be paralyzed.

Crutch paralysis is the term applied to that form of the disease which results from using poorly fitting crutches. It sometimes develops within a few hours after first using the crutches, sometimes after the lapse of days and weeks. The radial nerve is generally paralyzed on the side corresponding to the disease which necessitated the use of crutches. The paralysis sometimes occurs unexpectedly, sometimes it is preceded by paræsthesia. The ulnar and median nerves are often also paralyzed to a less extent. The radial nerve is affected mainly or exclusively because it is situated more posterially and inferiorly than the other nerves of the brachial plexus and is thus especially accessible to the pressure of the crutch.

Prisoner's paralysis is the term applied by Brenner to cases in which prisoners, whose arms are tied behind them, suffer from compression and paralysis of the radial nerves. Radial paralysis also occurs frequently in Russia in infants and coachmen; in the former, on account of the custom of tying the arms firmly to the chest and then laying the children on the side; in the latter, from the habit of tying the reins tightly around the arm.

Water carrier's paralysis is produced by carrying very heavy pails of water in such a manner that the arms are placed under the handle, and the pail rests upon the anterior surface of the chest and abdomen. The handle is, therefore, very apt to compress the radial nerve.

Traumatic radial paralyses also include cases which are the result of a blow, fall, incised or gunshot wounds, fracture or dislocation of the humerus. In such cases other nerves are also affected. In cases of fracture, the nerve may be directly injured by fragments of bone or by excessive growth of callus. This is most apt to occur in fracture of the lower third of the humerus.

II. ANATOMICAL CHANGES.—We are justified in assuming the same

conditions in the nerve and muscles that have been described in the section on facial paralysis. Bernhardt reported a case, after typhus fever, in which there was a neuritic swelling, three centimetres in length, in the course of the nerve. Above this point the nerve fibres were intact, below they were destroyed. The muscles innervated by the radial nerve presented a considerable proliferation of nuclei and indistinctness of the transverse striation.

III. Symptoms.—Peripheral paralysis of the radial nerve may be manifested by disturbances of motion and sensation. The former are more striking and constant, the latter may be entirely absent.

The following muscles are supplied by the radial nerve: triceps, supinator longus, supinator brevis, extensor digiti quinti proprius, ulnaris externus, anco-
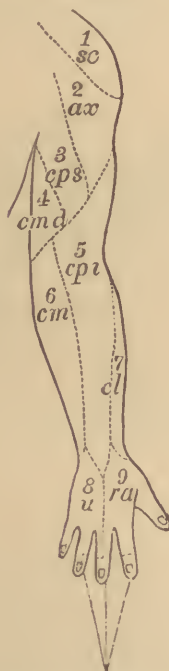
FIG. 10.   FIG. 11.
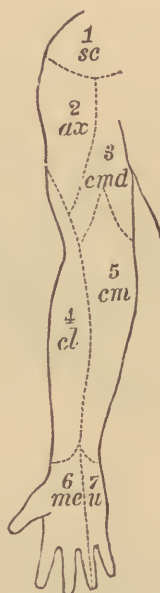
Distribution of the cutaneous branches of the radial and other nerves of the arms.

Fig. 10.—Dorsal surface of the upper limb. 1. *sc.*, supraclavicular nerves. 2. *ax.*, axillary nerve. 3. *cps.*, superior posterior cutaneous branch of the radial nerve. 4. *cmd.*, internal cutaneous nerve. 5. *cpi.*, posterior inferior cutaneous branch of the radial nerve. 6. *cm.*, greater internal cutaneous nerve. 7. *cl.*, lateral cutaneous nerve. 8. *u.*, ulnar nerve. 9. *ra.*, radial nerve. 10. *me.*, median nerve.

Fig. 11.—Volar surface of the upper limb. 1. *sc.*, supraclavicular nerves. 2. *ax.*, axillary nerve. 3. *cmd.*, internal cutaneous nerve. 4. *cl.*, external cutaneous nerve. 5. *cm.*, greater internal cutaneous nerve. 6. *me.*, median nerve. 7. *u.*, ulnar nerve.

næus quartus, abductor longus pollicis, extensor pollicis longus et brevis, extensor indicis proprius. The brachialis internus receives a branch from the radial nerve and also from the nervus perforans Gasserii.

The posterior superior cutaneous branch supplies the posterior surface of the arm as far as the elbow joint, the inferior posterior cutaneous nerve supplies the extensor aspect of the forearm as far as the wrist. The terminal

cutaneous filaments of the nerve supply the radial half of the dorsum of the hand, the entire dorsal surface of the thumb, the first and second phalanges of the index and middle fingers (vide Fig. 10 and 11).

The motor disturbances are easily recognized.   When the arm is held horizontally, the hand is kept in a position of volar flexion and slight pronation, the fingers are flexed, the thumb is flexed and bent under the fingers (vide Fig. 12).   The patients are unable to extend the hand and fingers; the grasp is feeble.   The fingers cannot be employed in delicate manipulations.   Supination of the forearm and, if the triceps is paralyzed, extension cannot be performed.

Dorsal flexion of the basal phalanges is rendered impossible on account of paralysis of the extensor digitor. communis.   Dorsal flexion of the middle and last phalanges is effected by the interossei (supplied by the ulnar nerve), but these only act when the basal phalanx is extended, so that the latter must first be passively extended before the other phalanges can be extended.   The feebleness of the grasp is also the result of paralysis of the common extensor, since the flexors

FIG. 12.

Position of the fingers and hand in peripheral radial paralysis in a man æt. 25 years.

can only act vigorously when the extensors also contract, and thus separate as much as possible the points of origin and insertion of the flexors.   The grasp becomes stronger as soon as the hand is passively extended.   For the same reason, adduction and abduction of the fingers are not performed properly until the hand and fingers are extended.   Paralysis of the ext. radial. longus et brevis and the ext. ulnaris is shown by the fact that adduction and abduction of the hand are interfered with or abolished.   If the arm is extended, supination of the forearm is not possible, because the supinator brevis is incapable of function.   If the forearm is flexed, supination may be effected by the biceps.   Paralysis of the triceps and anconæus is shown by the absence of resistance to passive flexion of the forearm.

Paræsthesiæ occur much more frequently in radial paralysis than sensory disturbances which are objectively demonstrable.   The patients complain of a feeling of coldness, numbness, and formication.   Since the majority of paralyses of the radial nerve originate at the point of flexion of the nerve, sensory disturbances are occasionally present only upon the dorsum of the hand.   Even if the lesion is situated higher, sensory dis-

turbances may be absent, because other nerves act vicariously for those in which conduction is interrupted.

Vaso-motor disturbances, viz., diminution of temperature and livid color of the skin, are occasionally observed.

Thickening of the joints of the fingers and hand, and nodular thickenings on the extensor tendons (tenosynitis hypertrophica of Gubler) may also occur, but their mode of origin is unknown. Atrophic changes develop in the muscles if the paralysis lasts a long time.

The electrical reactions of the nerve and muscles are the same, in general, as in peripheral facial paralysis. In "sleep" paralysis, the electrical irritability of the peripheral portion of the nerve is almost always retained, and often increased. Irritation of the central end produces no effect, so that the site of the lesion can thus be determined with accuracy.

In one case Fischer noticed increased mechanical irritability of the muscles.

The duration of the disease varies extremely. Crutch paralysis usu-

FIG. 13.
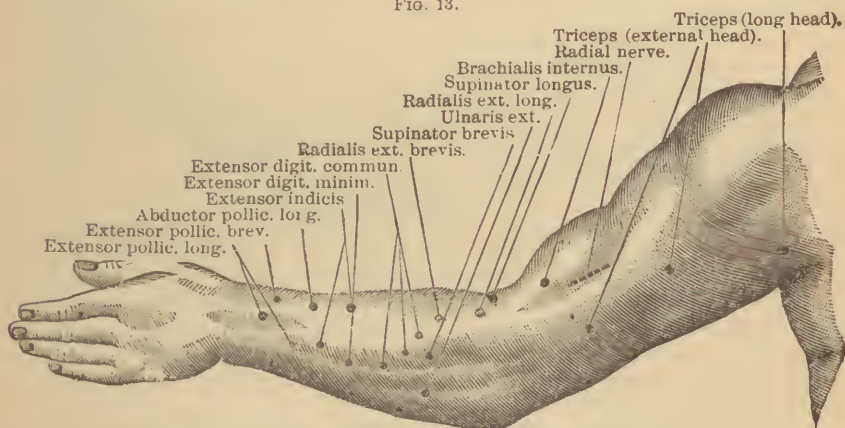
Motor points of the radial nerve and the muscles supplied by it.

ally recovers most rapidly. Sleep paralyses generally require four to six weeks before recovery is established.

IV. DIAGNOSIS.—The peripheral character of the paralysis is usually evident from the fact that the radial nerve alone is paralyzed. But Raynaud described a case in which a solitary tubercle in the fissure of Rolando at the origin of the second frontal convolution gave rise to isolated paralysis of the radial nerve. Apart from the rarity of such cases, the peripheral nature of the cause in this disease is usually evident from the previous history, the objective changes, and electrical examination. Lead palsy of the radial is usually bilateral, the supinators are almost always unaffected, and, as a rule, other symptoms of lead poisoning have been present. The situation of the lesion will be easily recognized from the number and grouping of the paralyzed muscles, the sensory disturbances, and particularly from the electrical examination of the trunk of the nerve.

V. PROGNOSIS AND TREATMENT.—The prognosis is usually good, and the hope of recovery should not be abandoned even if the paralysis has lasted a long time.

Treatment should first be directed toward the removal of the cause. In crutch paralysis better fitting crutches should be worn; dislocations must be replaced; if the nerve is compressed by callus, the latter must be removed and the nerve freed; if a cicatrix has formed in the nerve as the result of injury, it must be excised and the ends united with sutures, etc.

Local treatment consists exclusively of electrical applications. The motor points are shown in Fig. 13. Remak recommended the use of galvanism, the cathode upon the site of lesion, the anode upon an indifferent part (the sternum), current of moderate strength. The faradic current is preferable in applications to the muscles; three to four sittings weekly, each lasting three to five minutes.

6. *Paralysis of the Median Nerve.*

I. ETIOLOGY.—Peripheral paralysis of the median nerve is much rarer than that of the radial nerve. In the majority of cases it is traumatic, the nerve being injured, as a rule, in the forearm (not infrequently immediately above the wrist) by stab or incised wounds, etc. It is observed occasionally after unskilful venesection at the bend of the elbow. Injury by pieces of glass is relatively frequent.

Rheumatic paralysis of this nerve is rare. The disease is observed occasionally after acute infectious diseases (variola, typhoid), and also as the result of neuritis.

II. SYMPTOMS AND DIAGNOSIS.—If the lesion is situated immediately above the wrist, the paralysis is confined to the muscles of the ball of the thumb (abductor, flexor, and opponens pollicis brevis); the adductor pollicis brevis escapes because it is supplied by the ulnar nerve. In addition, there is paralysis of the first and second lumbricales. If the lesion is situated higher, there is also paralysis of the larger part of the volar muscles of the forearm, viz., pronator teres, flexor carpi radialis, palmaris longus, the superficial and deep flexors (these also receive branches from the ulnar nerves), flexor longus pollicis and pronator quadratus.

Paralysis of the flexor digitorum sublimis causes inability to flex the second phalanges; as the result of paralysis of the deep flexor, the terminal phalanx of the second, and often of the third finger cannot be flexed. The first phalanges are flexed by the interossei, which also extend the second and third phalanges (innervation from the ulnar nerve). Excessive contraction of the interossei sometimes produces hyperextension of the second and third phalanges, and a sort of subluxation. Volar flexion of the hand is impaired; it is effected by the ulnaris internus. If the forearm is extended, pronation cannot be performed; when flexed, it can be pronated imperfectly by the aid of the supinator longus.

Paralysis of the muscles of the ball of the thumb incapacitates the thumb from all delicate manipulations. Its phalanges cannot be flexed, and the movement of opposition cannot be effected. The non-paralyzed adductor pollicis (supplied by the ulnar nerve) draws the thumb against the index finger, and the extensors flex it strongly towards the dorsum.

Sensory disturbances may be entirely absent because the cutaneous branches of the ulnar and radial nerves vicariously perform the function of the median. In other cases there is loss of sensation on the radial half of the palm of the hand as far as the median line of the fourth finger, and on the dorsal surface upon the terminal phalanges of the

thumb and second and third fingers (vide Figs. 10 and 11).   The anæsthesia is often most marked on the tip of the index finger.

Trophic disturbances occur with relative frequency.   The affected fingers have a peculiar smooth and shining appearance (glossy skin), and the hairs are unusually long and abundant; the nails become thickened and distorted and exfoliate; pemphigus-like vesicles and ulcerations may form upon the terminal phalanges.

Atrophy of the muscles often develops after the paralysis has lasted a long time.

FIG. 14.
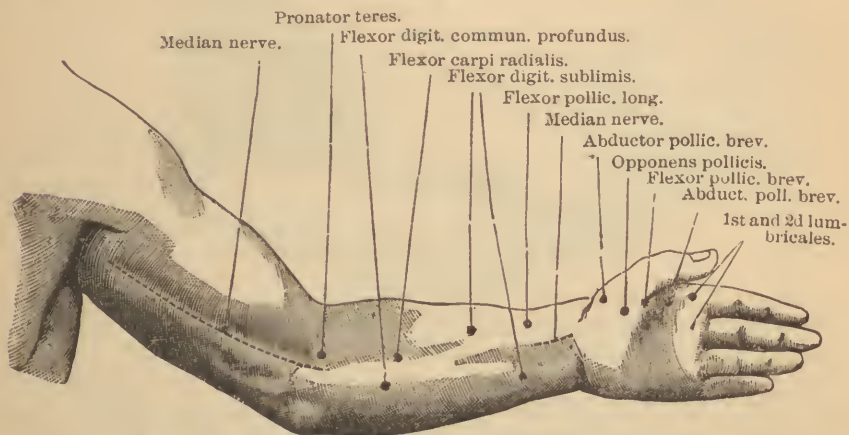
Motor points of the median nerve and the muscles supplied by it.

The electrical excitability of the nerve and muscles corresponds to that usually observed in peripheral paralysis.

III. PROGNOSIS AND TREATMENT are the same as in radial paralysis.

7. *Paralysis of the Ulnar Nerve.*

1. The most frequent cause of peripheral ulnar paralysis is traumatism: pressure of a crutch, fracture of the humerus and compressing

FIG. 15.
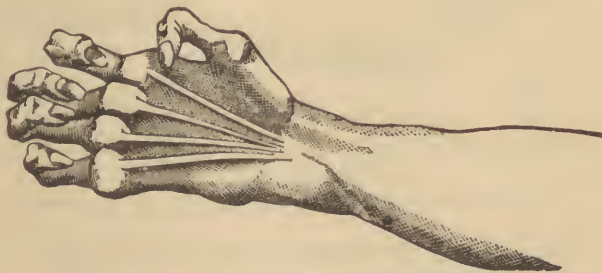
Claw-hand.   Palmar surface.   After Duchenne.

callus, tumors, gunshot or incised wounds, etc.   Compression paralysis sometimes develops after prolonged dorsal decubitus.   Duchenne observed it in laborers who are compelled to press the elbow for a long time against

a firm support.   Rheumatic or neuritic paralyses, and those occurring after infectious diseases, are much rarer.

2. The symptoms are readily interpreted. In paralysis of the muscles of the ball of the little finger (abductor, flexor brevis, opponens), the movements of the little finger are almost entirely abolished.   Paralysis of the interossei and third and fourth lumbricales causes impairment or

FIG. 16.

Claw-hand.   Dorsal surface.   After Duchenne.

abolition of the movements of adduction and abduction.   In addition, the first phalanges cannot be flexed, and the others cannot be extended (paralysis of the interossei).   As the ulnar nerve supplies the ulnar portion of the flexor profundus digitorum, flexion of the two or three outer fingers is impaired.   Paralysis of the adductor pollicis prevents active approximation of the thumb to the index finger.   Flexion and adduction

FIG. 17.

3d and 4th
  lumbricales.
Opponens digit.
    minim.
Flexor digit. minim.
Abduct. min. digit.
Palmaris brevis.
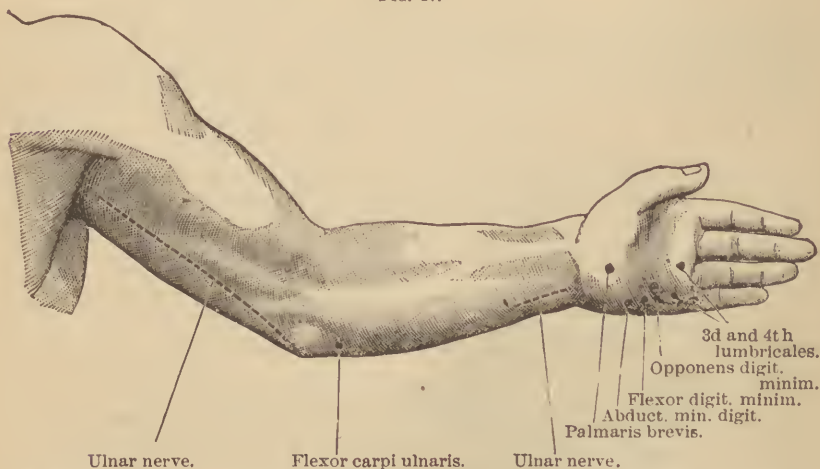
Ulnar nerve.   Flexor carpi ulnaris.   Ulnar nerve.
Motor points of the ulnar nerve and the muscles supplied by it.   Volar surface of the arm.

of the hand toward the ulnar side cannot be performed if the ulnaris internus has been paralyzed.

If cutaneous branches of the ulnar nerve are affected, sensation will be impaired upon the ulnar portion of the palm of the hand as far as the median line of the fourth finger.   Upon the dorsal aspect cutaneous sensibility is affected as far as the median line of the middle finger (vide

Figs. 10 and 11). Sensory disturbances may be entirely absent on account of the vicarious action of other nerves of the arm.

Trophic disturbances, on the whole, are rare. After long duration of the disease, muscular atrophy sets in, especially in the ball of the little finger and the interosseous spaces. The hand not infrequently assumes a peculiar shape, which is known as the claw hand. On account

FIG. 18.

Abductor digit. minim.
1st, 2d, 3d and 4th interossei.
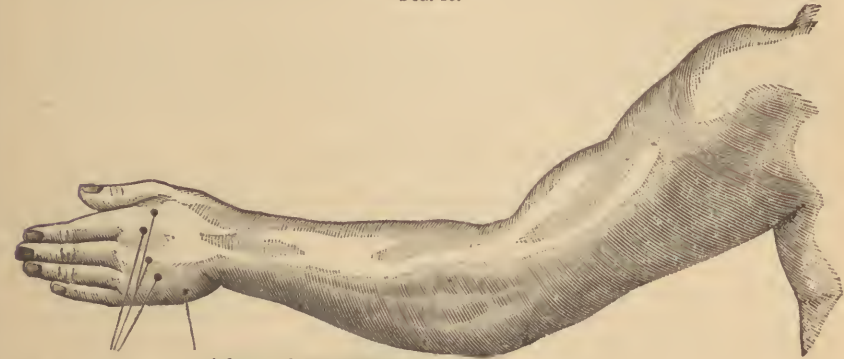
The same. Dorsal surface of the arm.

of the paralysis of the interossei and lumbricales, the extensor digitorum communis becomes excessively contracted, and the first phalanges are flexed so strongly towards the dorsum that they become slightly luxated. At the same time, the second and third phalanges are strongly flexed by the action of the flexors, so that the tips of the fingers are imbedded in the palm of the hand (vide Figs. 15 and 16). These changes

FIG. 19.

Musculo-cutaneous nerve.
Biceps.
Brachialis internus.
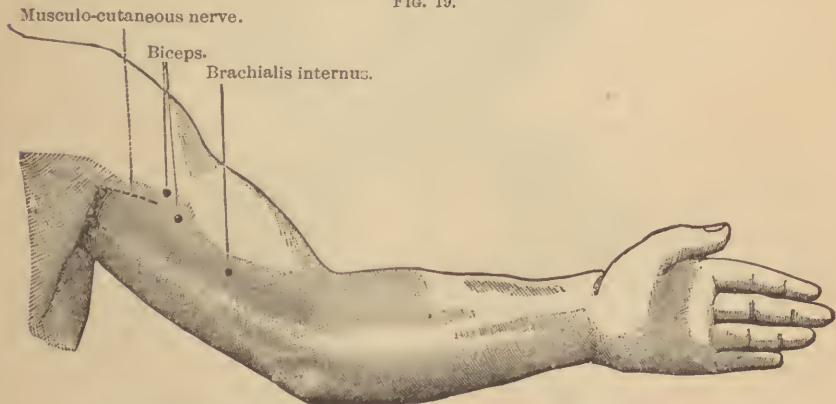
Motor points of the musculo-cutaneous nerve and the muscles supplied by it.

are especially marked in the fourth and fifth fingers, because the first and second lumbricales (second and third fingers) are supplied by the median nerve.

The electrical reactions are the same as in other peripheral paralyses.

3. The treatment is the same as that of radial paralysis. The motor points are shown in Figs. 17 and 18.

8. *Paralysis of the Musculo-Cutaneous Nerve.*

Isolated peripheral paralysis of this nerve is very rare, and it generally escapes even in combined paralysis of the nerves of the arm. Erb noticed isolated paralysis of the nerve after extirpation of a tumor in the supraclavicular fossa. The motor disturbances produced are paralysis of the biceps, coracobrachialis, and brachialis internus; but the latter muscle is also supplied by the radial nerve. The forearm can be flexed very imperfectly or not at all upon the arm. This is most marked in supination of the forearm; in pronation, the supinator longus aids in flexing the forearm. Any sensory disturbances which may be present are found upon the radial surface of the forearm (vide Figs. 10 and 11).

9. *Paralysis of the Axillary Nerve.*

Peripheral paralysis of the axillary nerve is most frequently of traumatic origin, but several cases of rheumatic paralysis have also been de-

FIG. 20.

Dorsalis scapulæ nerve . . . .

Axillary nerve . .

Long thoracic nerve (serratus magnus.

Phrenic nerve . . .

Ant. thoracic nerve (pectoral muscles)

Brachial plexus.

Erb's supraclavicular point.

scribed. Not infrequently it follows inflammation of the shoulder joint, perhaps because the inflammation extends to the axillary nerve and gives rise to neuritis.

The motor disturbances consist simply of loss of function of the deltoid muscle, so that the patient is unable to lift the arm to the hori-

zontal. The teres minor may be affected, but no noticeable symptoms are thereby produced. After paralysis of long standing, the deltoid undergoes atrophy, a deep horizontal furrow is felt and seen beneath the acromial end of the clavicle, and the head of the humerus is excessively movable in its socket. Sensory disturbances may be found upon the external and posterior surfaces of the arm (vide Figs. 10 and 11). The motor point is shown in Fig. 20.

10. *Combined Paralysis of the Nerves of the Arm.*

Peripheral paralysis of several nerves of the arm occurs not infrequently, but, as a rule, that of one nerve, particularly the radial, predominates. This form of disease is to be expected if the cause of paralysis is located in the brachial plexus. Seeligmueller claims that paralysis of the plexus is often associated with disturbances of the sympathetic, contraction of the palpebral fissure and pupil, and abnormal redness of the skin. From the previous remarks on paralysis of the individual nerves, we can readily determine what nerves are affected in each case.

Traumatism is an unusually frequent cause of combined paralysis of the nerves of the arm. This includes dislocation of the head of the humerus (especially subcoracoid dislocation), fracture of the humerus or clavicle, a blow or fall on the shoulder, more rarely incised or gunshot wounds in the lower part of the neck. The disease has also been observed as the result of the use of crutches, and of tumors in the neck.

A special form of disease is that in which the paralysis is confined to the deltoid, biceps, brachialis anticus and supinator longus, and occasionally involves the infraspinatus, supraspinatus, and supinator brevis. Erb showed that these muscles can be made to contract by placing one electrode of a faradic current alongside the transverse process of the sixth cervical vertebra about two to three centimetres above the clavicle, and a little behind the external border of the sterno-mastoid (vide Fig. 20). Hence the nerves supplying the muscles in question must be situated in close apposition at this locality (Erb's supraclavicular point). This form of paralysis may be the result of rheumatic causes, tumors in the neck, injury to the shoulder, and parturition.

Duchenne applied the term parturition paralysis to those cases of combined paralysis of the nerves of the arm occurring in the new-born as the result of difficult labor. The paralysis usually follows luxation or fracture of the humerus, fracture of the clavicle or scapula, more rarely direct compression of the plexus. These accidents may happen during version, application of the forceps, application of the hook, etc. It is doubtful whether the disease may occur during natural labor if the pelvis is narrow. The paralysis should not be mistaken for separation of the epiphysis of the humerus.

Combined paralysis of the nerves of the arm may also be the result of fractures and dislocations of the lower end of the humerus or radius, the application of tight bandages, etc.

11. *Peripheral Paralysis of the Nerves of the Scapula and Trunk.*

a. Peripheral Paralysis of the Serratus Magnus.

I. ETIOLOGY.—The serratus magnus is supplied by the long thoracic nerve. On account of the long and superficial course of the nerve, the muscle is frequently paralyzed.

Among the causes are rheumatic influences, and still more frequently traumatism (fall, blow, incised wounds, surgical operations, carrying heavy loads on the shoulder, etc.). In not a few cases the disease is the result of over-exertion of the muscle, for example, in sawing, mowing, etc. Secligmueller observed paralysis of the left serratus magnus in a feeble nurse who over-exerted herself in carrying a heavy child. It sometimes develops after infectious diseases, particularly typhoid fever, but also after diphtheria.

The right serratus is more frequently affected. The disease is much more frequent in males than in females; it is rare under the age of fifteen years.

II. SYMPTOMS.—The symptoms consist chiefly of changes in the position of the scapula, which are noticeable during repose, but are much more marked when the arm is raised. As a rule, they develop slowly, and are usually preceded by pains in the supraclavicular region and top of the shoulder (implication of cutaneous branches of the brachial plexus). After the paralysis appears, the primary neuralgia not infrequently gives way to cutaneous anæsthesia.

When the arm is dependent, the inner border of the scapula is more closely approximated to the spine than on the healthy side, and the lower angle of the bone is separated from the trunk to an unusual degree. At the same time the lower angle is nearer the spine than the superior internal angle, and accordingly the superior external angle is abnormally depressed. This is owing to the fact that, on account of the serratus paralysis, the trapezius, rhomboids, and levator anguli scapulæ assume the upper hand, and draw the scapula towards the spine. In the same way, the pectoralis major, biceps, and coraco-brachialis lift the lower angle of the bone away from the trunk. The contracted rhomboids and levator anguli scapulæ are not infrequently visible beneath the skin as more or less thick strands between the scapula and spinal column. If these muscles are also paralyzed, the change in the position of the scapula is lessened. Nor must it be forgotten that primary contracture of these muscles may produce the same position of the scapula as is seen in serratus paralysis, except that, in the latter, passive movements of the scapula are readily performed.

The changes become more marked when the arm is raised in front. The scapula then approaches the spine still more closely, and together with the lower angle of the bone is separated very markedly from the surface of the thorax (vide Fig. 21). The patients are unable to raise the arm slowly and steadily above the horizontal, because this movement requires rotation of the scapula by the serratus magnus. Elevation of the arm to the vertical position becomes possible if the scapula is pushed forwards passively, and its edges are pressed against the thorax. Some patients are able to raise the arm to the vertical by a swinging motion. The power of crossing the arm, or pushing it forwards, is also lost, since these movements are effected by the serratus. The patients are unable to offer any resistance to passive retraction of the scapula.

The electrical changes are similar to those observed in other peripheral paralyses. The motor points are shown in Fig. 20. The nerve is also readily reached in the axilla. If the muscle undergoes atrophy after paralysis of long standing, the digitations of the muscle, which are readily seen on the healthy side when the arm is raised, disappear and the lateral surface of the thorax is flattened.

b. *Paralysis of the Pectoralis Major and Minor.*—Both muscles are

supplied by the anterior thoracic nerves. They are very rarely paralyzed alone. Such an isolated paralysis would be shown by the impairment or abolition of abduction of the arm against the trunk, and inability to resist passive abduction of the arm. The patient is also unable to place the hand of the paralyzed side upon the opposite shoulder. If the muscles undergo atrophy, the infraclavicular region will be unusually flat. The motor points are shown in Fig. 20.

c. Paralysis of the Rhomboids and Levator Anguli Scapulæ.—These muscles are supplied by the dorsalis scapulæ nerve. Isolated paralysis of the muscles will produce no special symptoms. If the trapezius is also

Fig. 21.

Position of the scapula in paralysis of the serratus magnus, during elevation of the arm in front. After Duchenne.

paralyzed (vide page 16), the paralysis of the rhomboids is shown by the inability to approximate the scapula towards the spine, that of the levator anguli scapulæ by inability to move the shoulder upwards.

d. Paralysis of the Latissimus Dorsi.—Isolated paralysis of this muscle, which is supplied by the subscapular nerves, is rare. In this disease adduction of the arm against the trunk is diminished, the raised arm can be drawn downwards with less force, and the hand can with difficulty be carried towards the buttocks. Rotation of the arm internally

is very little or not at all impaired, since the subscapular and teres major muscles act vicariously in performing this movement.

e. Paralysis of the Subscapular and Teres Major (*internal rotators of the arm*).—These muscles are also supplied by the subscapular nerves. The predominance of the external rotators causes the arm to assume a false position, the volar surface of the hand being directed anteriorly and externally. Hence, manipulations of the hand upon the opposite half of the head and body are rendered difficult or impossible, and the patients are unable to rotate the arm internally.

f. Paralysis of the Infraspinatus and Teres Minor (*external rotators of the arm*).—This form of paralysis is more frequent than that of the internal rotators of the arm. The infraspinatus muscle is supplied by the subscapular nerve, the teres minor by the axillary nerve. If the arm is passively rotated internally, the patients are unable to rotate it outwards. If there is atrophy of the infraspinatus, the infraspinous fossa is abnormally flattened or depressed. The internal rotators generally give the arm a false position, the ulna looking forwards. Paralysis of the infraspinatus interferes with drawing, writing, and the use of the needle, since these manipulations necessitate external rotation of the arm.

g. Paralysis of the Extensors of the Back.—This disease is relatively frequent after typhoid fever; it is sometimes the result of traumatic or rheumatic causes.

In paralysis of the lumbar extensors, the chest of the patient, in standing, is bent over backwards, while the lumbar spine presents lordosis. While the patient is sitting, on the other hand, the lumbar spine presents kyphosis, which disappears during dorsal decubitus. The patient has a waddling gait. If he is sitting upon the floor, and endeavors to assume an erect position, he first makes rotary movements of the trunk, then grasps the thighs with the hands, and gradually, as it were, climbs up his own thighs. If the muscles have undergone atrophy, the loins are notably smaller.

In paralysis of the dorsal extensors, the spine presents paralytic kyphosis in the erect position, scoliosis if the paralysis is unilateral. These changes disappear in dorsal decubitus.

h. Paralysis of the abdominal muscles is very rare. If it is unilateral, the umbilicus will be drawn towards the healthy side during each expiration. In bilateral paralysis, the patients walk with the body bent over forwards; in addition, there is lordosis of the lumbar spine.

Expiratory movements (coughing, sneezing, micturition, defecation, etc.) are difficult or impossible. The abdomen appears distended.

The treatment of the various forms of paralysis mentioned includes the application of electricity, baths, massage, and gymnastic exercises. In hopeless cases orthopædic apparatus may be applied.

12. *Paralysis of the Phrenic Nerve.*

1. Paralysis of the diaphragm appears to be produced more frequently by direct lesions of the muscle than by diseases of the phrenic nerve. It is not uncommon in pleurisy and peritonitis, when the inflammatory process extends to the serous lining of the diaphragm. The trunk of the phrenic nerve is affected most frequently by tumors and abscesses in the neck. Rheumatic paralysis of the diaphragm has been observed in a number of cases. Whether the paralysis occurring in

lead poisoning and hysteria should be regarded as peripheral is more than doubtful. Oppolzer observed spontaneous paralysis of the diaphragm at the period of puberty.

In all these cases the paralysis sometimes affects the entire muscle, sometimes it is unilateral or even more circumscribed.

2. The symptoms are easily recognized, but it must be remembered that the respiratory disturbances may be absent during quiet breathing.

Upon exposing the patient, it is found that the epigastrium and the hypochondria are retracted during inspiration, and protrude during expiration. If the hand is introduced beneath the thorax, it is not elevated by the diaphragm during inspiration, and the lower border of the liver is found to ascend during inspiration and descend during expiration. Continued loud speaking or singing produces dyspnœa and exhaustion ; pressing movements are also rendered difficult. The patients get out of breath in walking, or going up-stairs, and the number of respirations may be increased to fifty per minute. Bronchial and pulmonary inflammations are extremely dangerous in this condition, because the power of coughing is impaired, so that dangerous congestion of the lungs and suffocation are apt to develop.

In one case Eulenburg noticed abolition of the reaction of the phrenic nerve to the faradic current. Electrical examination must be confined to the trunk of the nerve, which may be reached on the outer side of the sternomastoid, immediately above the omohyoid muscle (vide Fig. 20).

3. The prognosis is always grave. Treatment consists mainly of applications of electricity, in addition to removal of the causes. Both currents have been employed. One pole (cathode of constant current) is placed on the trunk of the nerve, the other on an indifferent spot (sternum, etc.), or on the hypochondrium. Contraction of the diaphragm is shown by protrusion of the epigastrium, and entrance of air into the air passages, accompanied by a movement of hiccough. Passage of the current transversely through the diaphragm is not attended with much success.

13. *Peripheral Paralysis of the Nerves of the Lower Limbs.*

Peripheral paralysis of the nerves of the lower limbs is rare; paralysis in this locality is usually spinal or cerebral in its origin.

a. Peripheral paralysis of the crural nerve has been observed in diseases and tumors of the lower part of the spine, hemorrhages near the cauda equina, inflammation of the psoas muscle and pelvic cellular tissue, tumors of the pelvis, dislocations and fractures of the femur, incised or gunshot wounds, after acute infectious diseases, etc. Paralysis of the quadriceps femoris has been observed not infrequently in connection with inflammation of the knee-joint.

Paralysis of the crural nerve is shown by motor disturbances of the iliopsoas, pectineus, sartorius, and quadriceps femoris. Paralysis of the iliopsoas muscle is shown by inability to flex the thigh upon the abdomen, that of the quadriceps femoris by inability to extend the flexed thigh. These muscles are also important in walking and in passing from the sitting to the erect position, so that these movements are seriously interfered with when the parts are paralyzed.

After paralysis of long standing, the muscles undergo atrophy, some-

times to such an extent that the anterior surface of the thigh seems to
be covered merely by the integument.

Sensory disturbances, when present, are found upon the anterior and
inner surface of the thigh and the inner surface of the leg (vide Figs. 22
and 23). If the cause of paralysis is situated very high, other sensory
nerves may also be affected. In paralysis of the external cutaneous
nerve, sensation is diminished or abolished upon the outer surface of the
thigh down to the region of the knee. Sensory disturbances on the
scrotum, lower part of the abdomen or loins must be attributed to

FIG. 22.   FIG. 23.
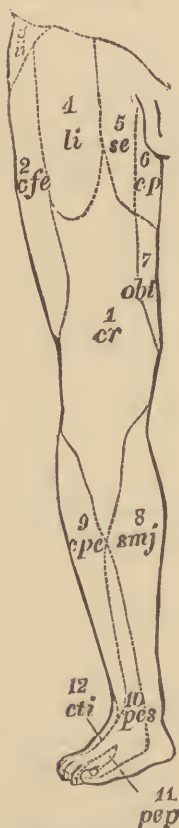
Distribution of the cutaneous nerves of the lower limbs. After Henle.

Fig. 22.—Anterior surface. 1, crural nerve; 2, external cutaneous nerve; 3, ilio-inguinal nerve; 4, lumbo-inguinal nerve; 5, external spermatic nerve; 6, posterior cutaneous nerve; 7, obturator nerve; 8, saphenus major nerve; 9, communicans peronei; 10, superficial peroneal nerve; 11, deep peroneal nerve; 12, communicans tibialis nerve.
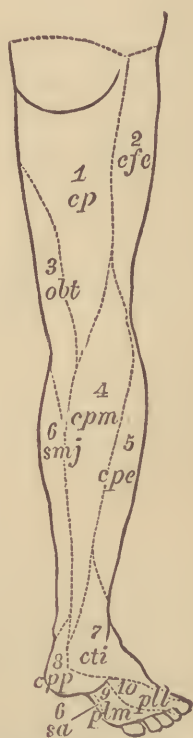
Fig. 23.—Posterior surface. 1, posterior cutaneous nerve; 2, external cutaneous nerve; 3, obturator nerve; 4, posterior mediau cutaneous nerve (peroneus nerve); 5, communicans peronei; 6, saphenus major nerve (crural nerve); 7, communicans ;tibialis; 8, nervus cutaneus plantaris proprius (tibialis); 9, plantaris medius (tibial nerve); 10, plantaris lateralis (tibial nerve).

implication of the ilio-hypogastric, ilio-inguinal, lumbo-inguinal, and
external spermatic nerves.

The electrical reactions present no changes from the general rules.

The nerve trunk can only be reached for a short distance immediately beneath Poupart's ligament, but the muscles are more readily accessible to electrical exploration and treatment (vide Fig. 24). In galvanic applications, the anode hould be applied to the loins, the cathode to the exit of the nerv immediately beneath Poupart's ligament.

b. *Periphera paralysis of the obturator nerve* is even rarer than that of the crural nerve; sometimes both nerves are paralyzed. Iso-

FIG. 24.
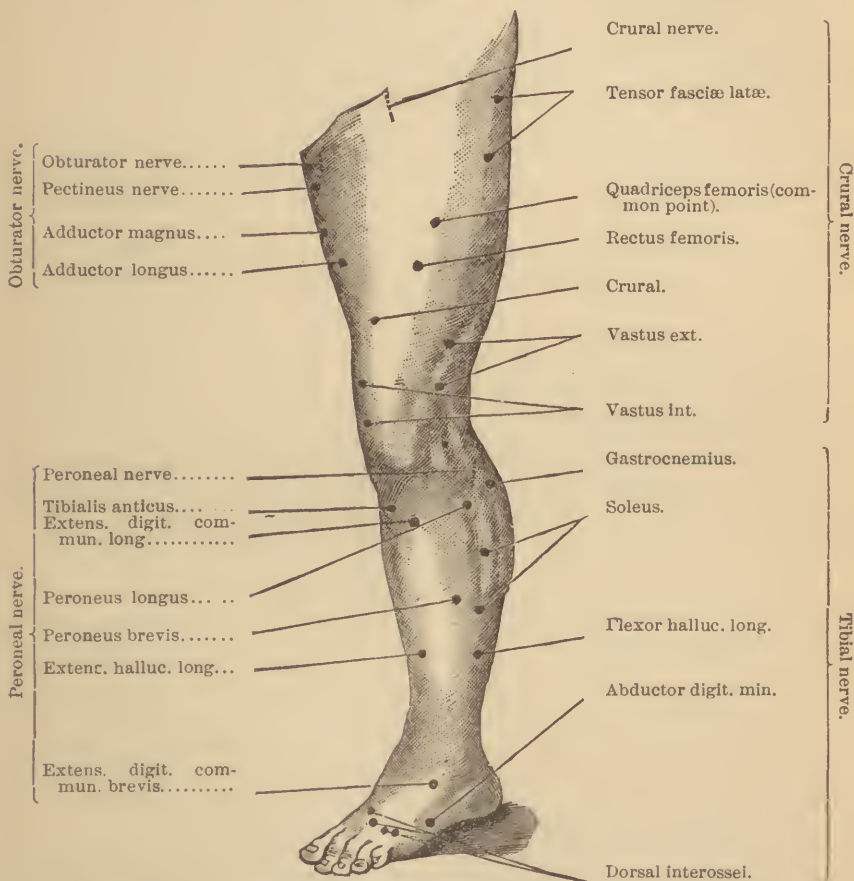
Motor points of the nerves and muscles of the lower limb on the anterior surface.

lated paralysis of the obturator nerve has been observed after difficult labor and in obturator hernia.

The motor paralysis affects all the adductors, the obturator externus, gracilis, and usually the pectineus, which is also supplied by the crural. The patients are unable to move the thigh towards the median line (paralysis of the adductors). In sitting or lying, they are unable to cross the affected leg over the healthy one, or to turn over from the back upon the belly, or vice versa. External rotation of the thigh is also interfered with.

3

Sensory disturbances are noticed upon the inner surface of the thigh as far down as the knee (vide Fig. 23).

c. Peripheral paralysis of the gluteal nerves is shown by motor disturbances in the gluteal muscles, the obturator internus, pyriformis, and tensor fasciæ latæ. There is impairment of internal and external rotation, and abduction of the thigh. The patient cannot pass from a bent-over forward position to an erect position of the body. Walking and ascending stairs are interfered with, because it is the function of the iliopsoas and gluteus maximus to fix the trunk upon the thigh and maintain equilibrium, but on account of paralysis of the gluteus the iliopsoas gains the upper hand and draws the trunk forward.

When atrophy of the muscles occurs, the buttock appears thin and flattened. The motor points are shown in Figs. 24 and 25.

d. Peripheral paralysis of the sciatic nerve is the most frequent form of paralysis of the nerves of the lower limbs. This is owing to the long and superficial course of the nerve. According to the location of the lesion, the paralysis affects the entire nerve or individual branches, particularly the peroneal or tibial nerves. The paralysis is sometimes confined to a few of the muscles supplied by the peroneal nerve.

Diseases of the lumbar spine, hemorrhages near the cauda equina, fractures of the pelvis or sacrum, fractures and dislocations of the thigh or leg, pelvic exudations and tumors, difficult labor, wounds, blows or falls in the region of the buttocks, removal of tumors, tight-bandaging, etc., are the chief traumatic causes of sciatic paralysis. It has been observed in the new-born after attempts at extraction. Zenker and Roth have described compression-paralysis of the peroneal nerve in potato-diggers who maintain a crouching or kneeling position for a long time. Sciatica is sometimes followed by paralysis of the nerve. Rheumatic paralysis is observed occasionally in individuals who are required to stand in cold water. Neuritic and hysterical paralyses of the sciatic have also been described.

The posterior muscles of the thigh supplied by the sciatic nerve are least frequently paralyzed, and only when the lesion is situated very high. The muscles in question are the obturator internus, gemelli, quadratus, semitendinosus, semimembranosus, long head of the biceps (the short head is supplied by the peroneal nerve). In paralysis of these muscles, rotation and adduction of the thigh are impeded, and the patient is unable to draw the leg against the posterior surface of the thigh, or to resist passive extension of the leg. The gastrocnemius (supplied by the tibial nerve) is able to effect slight flexion of the leg. As a matter of course, walking is interfered with to a considerable extent.

Paralysis of the peroneal nerve, if complete, affects the tibialis anticus, extensor hallucis longus, extensor communis longus, the peronei, extensor digitorum brevis, and extensor hallucis brevis. The disease is easily recognized. The foot assumes the varo-equinus position, and the patient is unable to flex the foot or the first phalanges of the toes towards the dorsal surface. During the attempt to walk, the foot hangs downward so that the toes are dragged along the floor unless the limb is unusually flexed at the hip joint. When the foot comes in contact with the ground, it first touches with the tips of the toes and the outer edge of the foot.

In isolated paralysis of the *tibialis anticus*, dorsal flexion and adduction of the foot are interfered with. Dorsal flexion is effected vicariously, in part by the extensor longus digitorum (the foot is abducted at

the same time); the extensor hallucis longus also aids the paralyzed muscle, at the same time adducting the foot. But these muscles cannot assume fully the function of the tibialis anticus, the calf-muscles acquire the predominance, and the foot is drawn into pes equinus position.

In isolated paralysis of the *extensor longus digitorum*, dorsal flexion and adduction of the foot are impeded. The first phalanx of the four outer toes cannot be extended. The function of the paralyzed muscle is performed vicariously by the tibialis anticus and extensor hallucis longus, but eventually the flexors undergo antagonistic contraction and the foot assumes pes varo-equinus position.

In isolated paralysis of the *peroneus longus*, adduction of the extended foot is impossible. At the same time the arch on the inner side of the foot is destroyed because the peroneus longus normally exerts traction on the cuneiform and scaphoid bones. Pes planus paralyticus is thus produced. In walking, the patient first brings the outer edge of the foot in contact with the floor; he complains of pain in the external malleolus, and is quickly tired.

In isolated paralysis of the *peroneus brevis*, pure adduction of the foot is impossible; this movement can only be effected if attended with dorsal flexion (contraction of extensor longus), or plantar flexion (contraction of peroneus longus).

Peripheral paralysis of the tibial nerve affects the following muscles: gastrocnemius, soleus, plantaris, popliteus, flexor longus digitorum, tibialis posticus, flexor hallucis longus, flexor brevis digitorum, abductor hallucis, flexor brevis hallucis, adductor hallucis, interossei, and lumbricales. If all these muscles are paralyzed, plantar flexion of the foot is almost entirely abolished (effected slightly by the peroneus longus); plantar flexion and adduction of the toes are impossible. Predominance of the extensors gives rise to pes valgo-calcaneus.

In isolated paralysis of the *gastrocnemius* and *soleus*, plantar flexion of the foot is markedly impaired; it is performed to a certain extent by the peroneus longus and flexor longus digitorum.

The calf-muscles also adduct the foot, so that when they are paralyzed the abductors also gain the upper hand. Hence pes calcaneus develops with a tendency to pes valgus.

Paralysis of the *tibialis posticus* interferes with adduction of the foot and elevation of its inner surface, *i. e.*, it favors the production of pes valgus.

Paralysis of the *flexor digitorum communis longus* is shown by inability to flex the second and third phalanges of the four outer toes. Flexion of the first phalanges is performed by the interossei, which also extend the second and third phalanges. In paralysis of the flexor hallucis longus, there is loss of plantar flexion of the last phalanx of the great toe. Paralysis of the interossei may give rise to the development of club-foot, from predominance of the extensor communis and flexor digitorum longus.

The electrical reactions of the paralyzed muscles are similar to those so often mentioned. More or less marked atrophy and contracture develop after paralyses of long standing.

Sensory disturbances are observed not infrequently; their distribution is shown in Figs. 22 and 23.

Trophic changes have been described, particularly in paralysis of the trunk of the sciatic nerve. They consist of coldness, cyanotic color of the integument, rapidly developing decubitus over the malleoli, heels,

buttocks, and trochanters, increased growth of hair, thickening of the nails, pemphigoid vesicles, etc. Paræsthesiæ have also been observed.

The diagnosis of paralysis of the sciatic nerve is not always easy; it must be based on accurate anatomical and physiological knowledge, strict analysis of the individual symptoms, and careful electrical exploration.

The prognosis is not always good; it depends on the causes of the disease and the results of electrical exploration.

The treatment consists chiefly in the application of electricity.

FIG. 25.
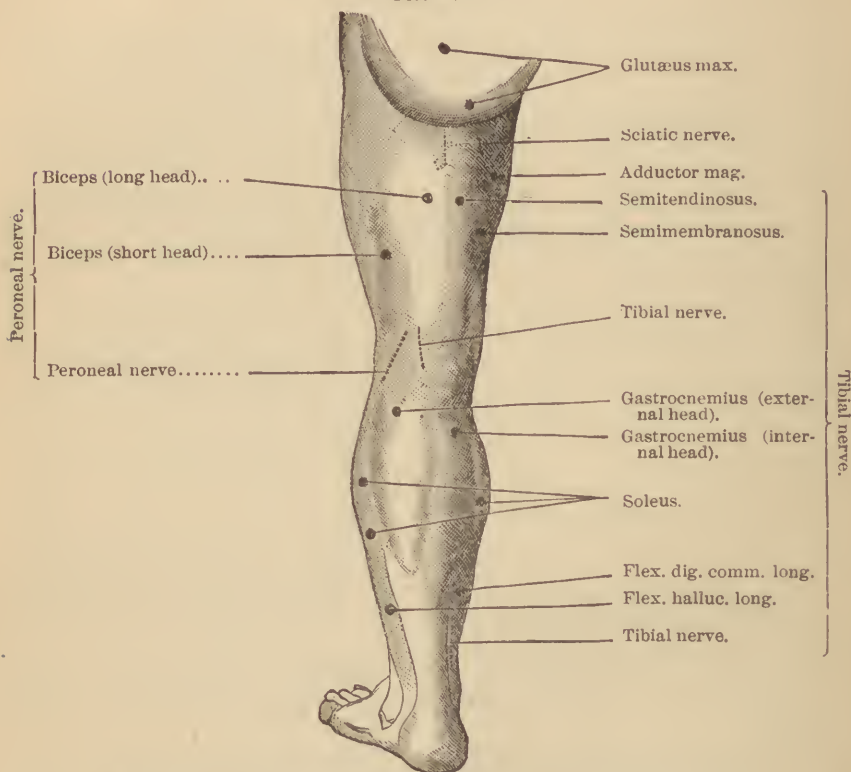
Motor points of the sciatic and its branches, the peroneal and tibial nerves.

The motor points are shown in Figs. 24 and 25. In hopeless cases, the use of orthopædic apparatus is indicated.

b. SPASM (HYPERKINESIS) OF THE MOTOR NERVES.

1. *Spasm in the Distribution of the Facial Nerve.*

(*Mimic Facial Spasm. Prosospasmus.*)

Spasms in the facial muscles are usually clonic, more rarely tonic. They may extend over the entire distribution of the facial nerve, or affect only individual muscles.

a. Diffuse Clonic Facial Spasm. (Tic convulsif.)

I. ETIOLOGY.—The disease occurs more frequently in males than in females, and is more common in advanced years than during childhood. It is sometimes the result of direct injuries to the facial nerve, sometimes it is a reflex effect of disturbances in other nerve tracts.

In certain cases, a cold has been mentioned as the immediate cause of the disease.

Other patients attribute it to injury. A number of cases have been reported in which the facial nerve was compressed or irritated by enlarged glands, diseases of the petrous portion of the temporal bone, exostoses, tumors at the base of the skull, aneurism of cerebral arteries, etc.

The reflex variety of facial spasm occurs most frequently in painful diseases in the distribution of the trigeminus (diseases of the lids and eyeball, painful dental affections, ulcerative processes in the nasal and buccal mucous membrane, neuralgia, etc.).

Mimic facial spasm also develops not infrequently in diseases of the genital apparatus, or irritation of the intestinal mucous membrane by worms. Moreover, painful pressure points are sometimes found on the spine, wrist-joints, and other parts of the body, treatment of which occasionally exerts a magical effect upon the reflex facial spasm.

There is no doubt that in some cases heredity plays an etiological part. But it must be remembered that the spasm is not always inherited as such, but that there is often merely a general neuropathic taint, manifested in the ancestors by hysteria, epilepsy, or other neuroses.

In some patients the nervous disposition is acquired, particularly during the course of chlorosis.

Mimicry is occasionally an etiological factor. Frequent "making faces" may finally terminate in well-marked facial spasm.

In a large number of cases no cause can be discovered.

II. ANATOMICAL CHANGES.—Nothing is known concerning the anatomical changes in this disease. The cases examined have given almost entirely negative results.

III. SYMPTOMS.—Mimic facial spasm consists of involuntary grimaces which occur in paroxysms. The patients suddenly wrinkle the brow, wink the lids, snuffle with the nose, and distort the whole face. The contractions are usually unilateral, rarely bilateral. They sometimes occur spontaneously, sometimes after physical or mental exertion. As a rule, they last only a few seconds; occasionally there are abortive attacks, in which a quick contraction passes rapidly over one side of the face. In some cases, only a few attacks occur daily; in others, twenty, thirty, or even more, in a single hour. Remissions and exacerbations are noticed, and depend chiefly upon the mental and physical condition of the patient. The twitchings generally cease during the night, but this rule presents exceptions. Some patients can voluntarily moderate the intensity of the contractions, but the will is powerless in the majority of cases. The palatal, digastric, and stylohyoid muscles are always, the auricular muscles and platysma myoides are usually, unaffected. The twitching may predominate in certain of the facial muscles, and this may vary in different attacks. In very violent and obstinate cases, the spasm may extend to other groups of muscles, viz., to the tongue, neck, and limbs.

The secretory and gustatory fibres of the facial nerve are unaffected.

In a number of cases, crackling, ringing, or roaring has been heard in the ear during the spasms (spasm of the stapedius?).

Pressure points should always be looked for.   These are points which correspond to individual sensory nerves, and are very sensitive to pressure; the latter may suddenly abolish, more rarely intensify, the spasmodic attack.   They may be found along the supra-orbital or infra-orbital nerve, on the mucous membrane of the nose or mouth, transverse or spinous processes of the cervical vertebræ, sternum, or intercostal spaces, wrist-joints, etc.

Tic convulsif is unattended with pain, but some patients complain of a peculiar tired feeling in the affected muscles.

The electrical reactions of the nerve and muscles are unchanged.

The disease lasts months and years; in many cases for life.

IV. DIAGNOSIS.—The recognition of the disease is easy, but it is often difficult to ascertain its cause.

V. PROGNOSIS.—The disease is never dangerous to life.   If the cause can be removed, the prognosis is usually favorable; if not, the disease is generally incurable.

VI. TREATMENT.—Causal treatment requires the most careful attention.   In some cases, the extraction of a carious tooth has effected a rapid and permanent cure.   In nervous individuals, we order nervines (potassium bromide, valerian, asafœtida, zinc, etc.); in chlorotic persons, iron preparations.   If the disease is the result of cold, resort should be had to the use of baths, diaphoresis, alcoholic frictions, leeches behind the ear, etc.

Pressure points should also be treated.   Applications of the galvanic current may be made, the cathode being placed on an indifferent spot (sternum, etc.), the anode upon the pressure point.   The current should not be too strong, and the sitting not prolonged beyond five minutes.

The same treatment may be employed if the tic is associated with trigeminal neuralgia.   If the latter is obstinate, neurotomy or neurectomy of the nerve affected by neuralgia sometimes proves successful.

If there are no causal indications, narcotics may be injected subcutaneously (℞ Magendie's solution, ¼ syringeful; atropine, gr. ⅙ : ℨ iij., ¼ syringeful; strychnine, gr. iss. : ℨ iij., ¼ syringeful; curare, gr. iss. : ℨ iij., ¼–½ syringeful).

As a rule, better results will be obtained from the use of electricity, although great patience is requisite.

The preference is generally given to the galvanic current. The anode is placed on the trunk of the nerve immediately in front of or behind the ear, the cathode upon an indifferent spot. The anode may also be applied stabile or labile to the individual muscles. Benedikt favors frequent changes of polarity. In diseases of the ear or base of the brain, the current may be passed transversely through the mastoid processes. In some cases, Erb and Berger advise the application of a large anode (head electrode) to the parietal bone, particularly when the disease is the result of central causes, in order to reach the cortical centre of the facial nerve. Remak recommends galvanization of the cervical sympathetic, the anode immediately behind the angle of the lower jaw to the outside of the carotid artery, the cathode on an indifferent spot. The same author also obtained good effects from descending currents, i. e., anode on the central, cathode on the peripheral portion of the nerve.

Frommhold recommended faradization of the muscles with currents of gradually increasing strength.

Various surgical operations have been performed to relieve the disease.   Thus, incision of the nerve and muscles has been performed.

Stretching of the nerve has been done with apparently good results, but Bernhardt showed that the effect is only temporary.

b. Diffuse Tonic Facial Spasm.

This disease is rare and is usually the result of previous facial paralysis; less frequently it is an independent, primary affection. The side of the face is stiff and rigid, the lines and grooves are unusually deep, the face appears drawn towards the affected side, the eyebrow is elevated; rigidity of the cheek sometimes interferes with mastication. The disease has been treated by galvanization of the contractured muscles, faradization of the healthy facial muscles, introduction of wooden globes under the cheek on the affected side.

c. Partial Tonic and Clonic Facial Spasm.

Contractions of individual facial muscles occur in many otherwise healthy persons as soon as they become excited. In others contractions occur during mental and physical rest, in the lips, alæ nasi, corrugator supercilii, frontalis, and even the auricular muscles. Contractions of the zygomatici cause a laughing expression (risus sardonicus). Rosenthal noticed isolated contractions of the palatal muscles.

Practical significance attaches to spasm of the orbicularis palpebrarum ; this is either tonic (blepharospasm) or clonic (nictitating spasm). The causes of both varieties are almost identical. In the majority of cases they result from diseases of the eye (diseases of the conjunctiva and cornea, foreign bodies, and other ocular affections attended with pain). A bright light will sometimes produce blepharospasm in otherwise healthy individuals. It may also occur in trigeminal neuralgia and diseases of the nasal, buccal, and palatal mucous membrane, and the teeth. As a matter of course, the spasm is the result in such cases of reflex processes which are conveyed from the sensory branches of the trigeminus to the facial nerve. The spasm may also be produced by irritation in other sensory tracts, for example, the uterine, intestinal nerves, etc. The causes of certain cases are unknown.

In blepharospasm the lids are suddenly and spasmodically closed; in some cases there is narrowing of the palpebral fissure. The spasm is almost always bilateral, lasts several seconds or minutes, even hours, and then ceases for a longer or shorter interval. In certain cases the spasm continues for weeks and months, so that the patient is constantly unable to see. The attacks often occur spontaneously, often as the result of bodily or mental excitement or ocular irritation. Apart from the deformity, the patients suffer from the fact that they may be suddenly rendered blind, in a mechanical manner, while walking the streets. The vigor of the contractions is usually so great that the lids cannot be forcibly opened. Towards the close of the seizure, its duration may sometimes be shortened by forcible separation of the lids.

Pressure points are sometimes found, pressure on which causes sudden cessation of the spasm. Graefe distinguishes primary and induced pressure points, the former are present from the beginning of the disease, the latter develop during its course.

These pressure points are observed most frequently along the supraorbital, next along the infraorbital nerve, and also on the mucous membrane of the nose, mouth, palatal arch, on the spine, wrist-joints, etc.

Blepharospasm sometimes gives rise to diffuse facial spasm, and even to spasm of more remote muscles, for example, on the back of the neck. Clonic spasm of the lids (nictitating spasm) is more frequent than blepharospasm, and in many individuals is merely a bad habit. It consists merely of winking movements. The remarks made concerning blepharospasm also hold good concerning this variety.

The prognosis of both affections is not always good; the disease is generally very obstinate, often incurable.

The treatment is the same as that of diffuse clonic facial spasm.

2. *Spasmodic Conditions of the Trigeminal Motor Branches.*

(*Spasm of the Muscles of Mastication.*)

I. The changes in question affect the temporal, masseter, and pterygoid muscles. The spasm of these muscles is sometimes tonic, sometimes clonic. In the former event, the muscular contractions recur so rapidly as to produce the impression of a permanent muscular contraction; in clonic spasm, the individual contractions can be distinctly distinguished from one another.

In the majority of cases, the spasms are the result of central changes, not often of peripheral lesions. Exposure, injury to the nerve, and trigeminal neuralgia have been mentioned as causes. A relatively frequent cause are diseases of the teeth (eruption of the molar and wisdom teeth), inflammations of the periosteum of the inferior maxilla or temporo-maxillary joint.

Reflex spasms occur in children suffering from worms, after peripheral injury, and painful cicatrices.

2. Spasm of the muscles of mastication is almost always bilateral. In tonic spasms, the contracted masseters and temporals protrude beneath the skin, and feel as hard as a board. The patients have very little or no power of drawing the lower jaw downwards or from side to side. As a rule, the mouth cannot be possibly opened by any amount of force which can be used with safety. This condition is known as trismus. Speech is difficult, and the patients are sometimes unable to take even fluid food into the mouth. Such conditions may last days and even weeks.

The best known form of clonic spasm of the muscles of mastication is the chattering of the teeth, which occurs when the individual feels very cold; the movements then occur from above downwards. In some cases, lateral movements are produced (grinding of the teeth). The tongue, gums, and mucous membrane of the mouth may be thereby injured.

3. Nothing is known concerning the anatomical changes.

4. The diagnosis is almost always easy. Tonic spasm is distinguished from ankylosis of the temporo-maxillary joint by the absence of the hard contraction of the muscles; in addition, the history of the disease is different. In doubtful cases, the patient may be anæsthetized with chloroform, whereupon the muscular spasm will subside, while the ankylosis persists.

5. The prognosis depends upon the etiology.

6. Treatment should first be directed towards the removal of the causes. A subcutaneous injection of morphine into the cheek seems to be the most reliable method of relieving the spasm. Chloroform may be used in very violent and obstinate cases. Electricity may also be

employed—the galvanic current transversely through the masseters, at first with feeble currents, then gradually increased, and as gradually diminished. Benedikt recommends frequent changes of polarity. Strong faradic currents, especially the wire brush, afford good results in some cases. In chronic cases, the attempt has been made to gradually relieve the trismus by the introduction of wooden wedges of gradually increasing size between the rows of teeth. It may be necessary to nourish the patient by means of the œsophageal sound, through the nose, or by means of nutritive enemata.

3. *Spasm in the Distribution of the Hypoglossal Nerve.*

Spasmodic conditions in the hypoglossal tract are shown by abnormal movements of the tongue; they are rare, more frequently central than peripheral, and usually have a clonic, more rarely a tonic character. We must distinguish between masticatory and articulatory hypoglossal spasms. The latter have been called aphthongia. This consists of attacks in which the tongue is moved involuntarily in all directions. In one of my cases, the tongue had been pushed against the teeth with such force that losses of substance occurred on the surface of the tongue. As a matter of course, speech and mastication are interfered with during the attacks. They occur in anæmic, nervous, and hysterical individuals, sometimes during trigeminal neuralgia or mimic facial spasm. The hypoglossal spasm is sometimes primary, and the facial spasm develops later. Sensory disturbances have been observed upon the mucous membrane of the tongue, lips, and mouth. The treatment is similar to that of facial spasm.

4. *Spasm in the Distribution of the Spinal Accessory.*

1. Spasm in the distribution of the spinal accessory affects the sternomastoid and trapezius muscles. The spasm may be tonic or clonic, unilateral or bilateral, and affect one or both of the muscles mentioned.

Some cases are undoubtedly rheumatic, others are traumatic in their origin; they may arise from over-exertion or lifting heavy weights. It is sometimes observed after infectious diseases (typhoid, puerperal fever, etc.). Or it may be the result of diseases of the spine (tuberculosis, tumors, fractures, etc.). Reflex influences can sometimes be demonstrated, for example, teething in childhood, helminthiasis, uterine diseases, etc. It is not infrequently impossible to ascertain the cause, and many cases of supposed peripheral diseases are undoubtedly central in their origin. This is true of nervous and hysterical individuals in whom spasm of the spinal accessory may alternate with or be followed by epilepsy, idiocy, or insanity. Central causes are directly demonstrable in certain cases.

2. In unilateral clonic spasm of the sternomastoid, the head is turned at each muscular contraction in such a manner that the chin looks toward the healthy side, and the mastoid process and lobe of the ear on the affected side are approximated to the clavicle. The contracted muscle is visible under the skin as a tense strand.

If clonic spasm of the trapezius is present at the same time, the head is drawn further backwards and the shoulder upwards, so that the occiput and shoulder are sometimes brought in contact. In certain cases, the contraction is irregularly distributed in the trapezius, so that more

or less torsion of the scapula results. The contractions of the sterno-mastoid and trapezius may alternate with one another, the vigor of one may predominate over that of the other, or both may be synchronous and equal in power.

In clonic spasm of both sternomastoids and trapezii, the symptoms vary according as the spasm occurs synchronously or alternately on the two sides. In the former event, the head performs nodding movements, but in the majority of such cases, other muscles of the neck are also implicated. This form occurs almost exclusively in children from the period of dentition to that of puberty. If the contractions occur alternately on the two sides, the head is wagged to and fro. The spasm is sometimes unilateral at first, and later spreads to the other side.

The attacks occur spontaneously, or they are produced by bodily or mental excitement. They usually cease during sleep, but many patients complain that they interfere with falling asleep. The number and duration of the attacks are subject to great variation. Many patients complain of a peculiar feeling of tension or exhaustion in the affected muscles, which may become intensified into severe pain. Pressure points are not frequent. Other groups of muscles often take part in the spasm, for example, those of the eyes, face, throat, shoulder, limbs, and in rare cases even general convulsions are produced. Romberg described œdema and paræsthesia of the arms as the result of pressure on the vessels and nerves in implication of the scaleni. The disease may last weeks, months, years, even a lifetime.

Tonic spasms of the sternomastoid and trapezius muscles produce permanent abnormal positions of the head (caput obstipum spasticum). The position which will be assumed by the head, according as one or both muscles are affected unilaterally or bilaterally, is evident from previous considerations.

The condition is sometimes congenital, or develops in early childhood, and thus often gives rise to secondary changes in the spinal column. The spine presents a convex curve toward the healthy side, the individual vertebræ appear smaller on the diseased side, and even the growth of the face may be retarded. The tonically contracted muscles are often hypertrophied, their antagonists atrophied.

3. The PROGNOSIS of tonic and clonic spasms of the spinal accessory nerve is not always good. As a rule, there is no chance of recovery unless the cause can be removed. Many patients have been driven to suicide by their obstinate disease.

4. TREATMENT must first be directed to the removal of the cause of the disease. The chief local method of treatment, apart from subcutaneous injections of morphine, curare, atropine, or strychnine, is the application of electricity—constant stabile current, anode to the affected nerve or muscle. Busch obtained very rapid and successful results from the use of the actual cautery. Orthopædic apparatus and gymnastic exercises may become requisite. Excision of the nerve and myotomy of the muscles are hardly justifiable.

5. *Spasm of the Neck, Scapula, and Arm.*

These muscles are supplied by the cervical and brachial plexuses. The spasms may be tonic or clonic, unilateral or bilateral, and affect a single muscle or various groups of muscles. We will refer to only a

few varieties. The etiology, prognosis, and treatment are the same as in spasm of the spinal accessory nerve.

a. Spasm of the obliquus capitis inferior.—Contraction of this muscle simply rotates the head in a horizontal line. In unilateral clonic spasm there are constant rotatory movements towards the affected side; in bilateral spasm there is rotation in both directions (rotatory tic). In fixation of an object the patients must hold the head between the hands in order to prevent the movements. In tonic spasm the head assumes a constant abnormal position to one side.

b. Spasm of the splenius capitis.—This muscle draws the head backwards; at the same time the face turns towards the contractured side, the chin is depressed and approaches the shoulder on the same side. The contracted muscle projects beneath the anterior border of the trapezius, the sternomastoid appears flaccid on the affected side, tense

Fig. 26.
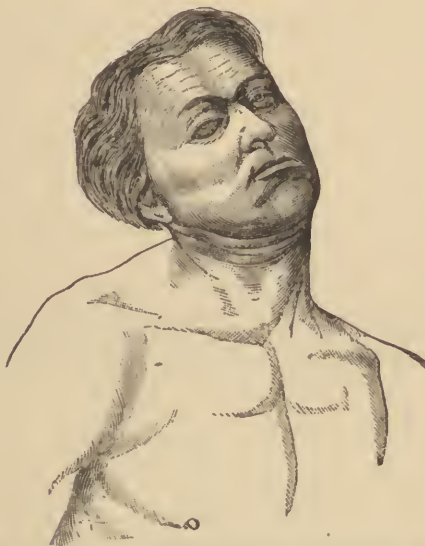
Position of the head in spasm of the right splenius capitis muscle.

upon the other side. Tonic spasm of the muscle is much more frequent than the clonic form.

c. Spasm of the rhomboid has hitherto been observed only in the tonic form. On contraction of this muscle, the lower angle of the scapula is elevated, its inner edge approaches the spine, and its direction changed so that it runs from below internally, upwards externally. The contracted muscle can be felt, and often seen in the interscapular space. Elevation of the arm to the vertical position meets with resistance.

d. Spasm of the levator anguli scapulæ produces elevation of the scapula, especially its upper inner angle, and bends the head backwards. The contracted muscle is visible in the supraclavicular fossa.

6. *Spasm of the Respiratory Muscles.*

a. Clonic spasm of the diaphragm is called hiccough, singultus. Upon sudden contraction of the diaphragm in this condition, the in-

spiratory current of air enters the air passages with a loud noise, but is suddenly checked by closure of the rima glottidis. The ailment is more frequently central than peripheral in its origin, and is especially frequent in nurslings. In rare cases it is the result of irritation of the trunk of the phrenic nerve by mediastinal tumors, aneurisms, pericarditis and pleurisy. Singultus sometimes occurs in diaphragmatic pleurisy. It develops more frequently in a reflex manner, for example, in overloading of the stomach, ingestion of too hot or too cold food, diseases of the intestines or liver, biliary or renal calculi, uterine diseases and peritonitis. It is especially noteworthy that prostatic diseases give rise not infrequently to hiccough.

Among the forms produced by central causes are included those cases which occur in diseases of the brain and meninges, in hysterical and anæmic individuals, during severe diseases, for example, after losses of blood, cholera, dysentery, cancerous cachexia, malaria.

The attacks may be so severe that sixty to eighty contractions of the diaphragm occur in a minute. The patients then suffer from dyspnœa, talking and eating are interfered with, and pain is felt in the epigastrium and insertion of the diaphragm. Pressure points are sometimes noticed in remote parts. The attacks may last hours, days, weeks, even months, sometimes almost uninterruptedly, sometimes with free intervals. They usually cease at night, but interfere with falling asleep.

The disease sometimes resists all methods of treatment. We should endeavor to remove the causes and divert the attention of the patient. In several of my cases, rapid recovery was produced by constantly counting aloud at a uniform rate of rapidity. Vigorous straining, with closed rima glottidis, passage of the œsophageal sound, or injection of cold water into the œsophagus until suffocation threatens, have also been recommended. It is best to employ narcotics subcutaneously in the region of the diaphragm. In a colleague, æt. 72 years, I obtained rapid success from the hourly administration of ℞ Potassium bromide, gr. viiss.; ext. belladonnæ, gr. ¼. Vigorous cutaneous irritants—sinapisms or the faradic current—to the diaphragmatic region have also been tried. Recovery has been effected in some cases by galvanic or faradic treatment of the phrenic nerve. Finally, the lower part of the thorax has been compressed and the head firmly pressed against the chest a number of times, in order to produce vigorous expiratory movements.

b. Tonic spasm of the diaphragm, as the result of peripheral causes, has been observed after a severe cold or after intercostal neuralgia, muscular and articular rheumatism. As the result of central disturbances of innervation, it has been observed in tetanus, epilepsy, and hysteria. The disease is extremely dangerous, and will inevitably result in death unless speedily relieved.

The symptoms are easily recognized. The lowermost part of the thorax appears very dilated, but takes no part in the respiratory movements. The lower border of the right lung, and occasionally the heart, are lower than normal. The epigastrium is very prominent, and the normal respiratory movements of the diaphragm cannot be felt on palpation of the hypochondria. The upper part of the chest moves vigorously. The patient complains of dread of suffocation, has a cyanotic appearance, a small, hurried pulse, and is hardly able to talk in a loud tone. If rapid relief is not brought, death occurs from suffocation.

The treatment consists of the application of strong cutaneous irri-

tants—hot compresses to the region of the diaphragm, mustard poultices, the faradic brush.   We may give a subcutaneous injection of morphine or chloroform by inhalation; in addition, faradization or galvanization of the phrenic nerve.

APPENDIX.

Numerous other spasmodic conditions of the respiratory muscles have been described, but their combinations are so manifold that they cannot be described from a general standpoint.   This group includes spasms of sneezing (ptarmus s. sternutatio convulsiva), of yawning, crying, laughing, shouting.   As a rule, the causes are central, as in hysteria and organic diseases of the nervous system, or the result of reflex irritation.

7. *Spasm of the Abdominal Muscles.*

Westphal reported a case of clonic spasm of the abdominal muscles, which was relieved by the actual cautery.   I observed a similar case in an hysterical boy of nine years.   Tonic spasms of central origin occur in meningitis, tetanus, etc.

8. *Spasm in the Muscles of the Lower Limbs.*

Spasms rarely occur in the muscles of the lower limbs.   They may be tonic or clonic, and sometimes extend to all the muscles of the lower limbs.   We cannot enter into a detailed description, but, with regard to the function of the various muscles, refer the reader to the section on paralysis.

9. *Cramp.*

1. Cramp is a term applied to a tonic muscular spasm associated with pain.   It often lasts only a few seconds, and at all events, its duration is very brief.

The best known form is spasm of the calves of the legs.   This is a painful, tonic contraction of the muscles, which project with sharp contours, and are very tender on pressure.   Nevertheless, the muscles are not contracted ad maximum.   After the muscles are relaxed, at the end of a few seconds or minutes, a peculiar feeling of tension and exhaustion not infrequently is left over, and the muscles remain for a long time very tender on pressure. Ecchymoses are sometimes formed during the cramp. It is especially apt to occur at night, sometimes recurs very rapidly, disturbs sleep, or produces such violent pain as to give rise to syncope.

Cramps also develop in other muscles, but particularly in the lower limbs.

Schultz claims to have found increased electrical excitability of the affected muscles.

2. Among the causes of such conditions may be mentioned: *a*. Overexertion of the muscles, for example, cramps in the calves after long walks, dancing, etc.; *b*. Nutritive disturbances, for example, in cholera, as the result of losses of fluid (according to Erb, this also occurs in diabetes mellitus); *c*. Circulatory stasis, for example, the cramps in the calves in varicose veins and pregnancy.

3. Treatment consists of rest on the back (although many persons are relieved by hyperextension of the muscles), subcutaneous injections of morphine, friction or kneading of the muscles, inunctions of alcoholic solutions.

B. DISEASES OF THE SENSORY NERVES.

a. NEURALGIAS.

1. *Trigeminal Neuralgia.*

(*Facial Neuralgia, Prosopalgia, Tic Douloureux.*)

1. ETIOLOGY.—Among all the forms of neuralgia, that of the trigeminus is the most frequently observed. This is owing to its long course, the situation of many branches in narrow, bony canals, and the superficial position of its terminal ramifications.

It is most frequent in women, and from the twentieth to fiftieth years. It is very rare in childhood, more frequent in old age. The processes of the period of involution, *i. e.*, the fortieth year, on the average, in males, the menopause in female, favor the development of the disease. It is said to be more frequent in cold than in warm climates. According to some writers, individuals belonging to the well-to-do classes are predisposed to the affection. With regard to Goettingen, I may state that it is very common among the laboring classes.

The causes of the disease may be divided into five groups, constitutional, infectious, toxic, local, and reflex.

Hereditary factors are active in many cases. Sometimes we observe families in which several generations have suffered from trigeminal neuralgia, sometimes there is an hereditary nervous taint, so that different members of the family suffer from hysteria, epilepsy, psychopathies, or neuralgias. In some individuals the nervous disposition is acquired as the result of excesses, mental and bodily over-exertion, worry. Trigeminal neuralgia develops not infrequently in anæmia and chlorosis, which have been produced by vital losses, such as occur after protracted diarrhœa, rapidly following pregnancies, and excessive lactation.

Among the infectious forms the most important are those which are the result of malaria. In such cases the supraorbital nerve is almost always affected. The neuralgic attacks generally occur daily at the same time, more rarely they present a tertian or quartan type. But we have seen many typical cases of intermittent supraorbital neuralgia in individuals who were entirely free from malaria. The disease may also occur in typhoid fever and recent syphilis. In the majority of cases, however, syphilitic trigeminal neuralgia is the result of exostoses, gummata, inflammation, etc., at the base of the skull or within the bony canals.

Toxic trigeminal neuralgia has been observed in lead and mercurial poisoning.

The disease is very often the result of direct injury to the nerve, for example, as the result of cold, injury, inflammation of the periosteum, unskilful extraction of the teeth, caries and exostoses of the teeth, eruption of the wisdom teeth. Gross has called attention to obstinate neuralgia in toothless old people in whom the empty alveoli are gradually filled with bony substance, which irritates the alveolar nerves.

Trigeminal neuralgia may be the result of diseases of the ear, inflammation of the frontal sinuses with retention of secretion, and straining of the eyes.

The causes of the neuralgia must often be sought in the narrow bony canals through which the branches of the nerve pass. Compression and irritation of the nerve may be brought about by inflammation, thickening, and deposits in these localities, in many cases by simple distention of the blood-vessels. This danger is so much greater the narrower the bony canal, and the more numerous the blood-vessels situated within it.

The causes of neuralgia are sometimes found at the base of the skull, for example, inflammations, exostoses, tumors or aneurisms which press upon the trunk of the trigeminus.

Reflex trigeminal neuralgias include those which occur in uterine, ovarian, and intestinal diseases. The causal connection cannot be doubted in those cases

in which the neuralgia disappears as soon as the primary disease is relieved. Trigeminal neuralgia sometimes develops after injury of remote nerve tracts.

II. ANATOMICAL CHANGES.—Little is known concerning the anatomical changes in trigeminal neuralgia. Unusual redness and succulence of the affected nerve branches have been observed in some cases; in others, proliferations or calcification of the neurilemma, fatty and atrophic changes in the nerve-fibres or cells of the Gasserian ganglion. In Cruveilhier's celebrated case, nodules of carcinoma were found in the neurilemma of the peripheral branches of the facial nerve and the inosculating trigeminal fibres.

III. SYMPTOMS.—Trigeminal neuralgia is almost always unilateral, and, according to Canstatt, is more frequent on the right side (?). Bilateral neuralgia is extremely rare, and occurs only in the supraorbital nerve. It is equally rare that the neuralgia affects first one, then the other trigeminus. In a larger number of cases the disease spreads from one branch to a second or third.

The ophthalmic nerve is affected most frequently, particularly the supraorbital branch. Next in order of frequency is supramaxillary neuralgia, that of the infraorbital nerve being the most frequent in this class. The inframaxillary branch is least frequently affected; neuralgia of the mental or alveolar branches is the most frequent.

The entire nerve is rarely affected, and then only when the neuralgia is the result of intracranial causes, or of lesions affecting the trunk of the nerve. It is also rare to find all the ramifications of a single branch affected. The more the neuralgia is confined to the finer twigs, the more we are justified in assuming peripheral causes. Some branches exhibit very little tendency to neuralgia; this is particularly true of the auriculo-temporal nerve.

The neuralgic attacks not infrequently develop suddenly, in other cases they are preceded by prodromata. The latter almost always consist of paræsthesiæ : a feeling of stiffness, pricking, formication in the affected parts.

The neuralgia itself is characterized by attacks of pain, which sometimes have a boring or burning, sometimes a lancinating character. Some patients experience a sensation as if the nerve were being drawn out slowly, or as if the bones were slowly crushed. The pain is sometimes situated deeply, sometimes superficially. Some patients state that the pains radiate from the centre towards the periphery; much more rarely they pursue an opposite course. All patients agree that the pains are atrocious, and render them incapable of doing anything, so that very few are able to keep on with their occupation. The pain always extends along certain tracts of the nerve. In some cases it radiates into more remote parts, such as the back of the neck, and the extremities.

The attack usually lasts only a few seconds; in abortive attacks a single, lightning-like spasm of pain is experienced.

The number of attacks within an hour may be very considerable. In other cases attacks of variable duration occur at irregular intervals during the day. In intermittent neuralgia the attack recurs at a definite hour in the day, usually in the morning, or at noon, and disappears at the end of a certain time. In very rare cases it begins with a chill and ends in a sweat.

In many cases no immediate cause of the individual attacks can be demonstrated. In others they occur after exposure to a draught, on slight contact with the integument of the face, upon touching certain definite points, eating hard or cold articles of food, etc. Some patients

suffer after mental or physical excitement, if the eye is annoyed by a bright light, or the ear by shrill tones, on yawning, sneezing, laughing, often at the mere thought of an attack.

In the interparoxysmal period many patients feel entirely free from pain, others are tormented by more or less violent painful sensations.

Pressure points constitute a frequent, though by no means constant symptom. These are points which are situated in the affected nerve tracts, and are either the only parts which are painful on pressure or pain more than other parts. These points are sometimes present during the attack alone, sometimes during the interparoxysmal period. Pressure upon them may provoke a neuralgic paroxysm; strong pressure sometimes relieves the pain, while gentle pressure increases it.

Trousseau showed that pressure points are sometimes found upon the spinous processes of the second and third cervical vertebræ, or the external protuberance of the occipital bone.

Vaso motor disturbances are very often present during the attack. The affected half of the face, or a circumscribed nerve tract is very red, the arteries, especially the temporals, are unusually dilated and pulsate vigorously, and the cutaneous veins are also dilated. The skin has a puffy, peculiar shining appearance, is unusually warm, and covered with perspiration. The beginning of the attack is preceded by anæmia of the skin.

The conjunctiva is often injected, and this may even terminate in œdema (chemosis). The lachrymal secretion is increased, the eyeball seems to protrude from its socket.

The nasal mucous membrane secretes an increased amount of mucus, which is sometimes tinged with blood. In rarer cases the secretion of mucus is diminished.

There is sometimes an increased secretion of saliva, and the gums may present swelling, aphthous excoriations, and hemorrhages. In rare cases the patients complain of perverse gustatory sensations or auditory disturbances.

The trophic changes are closely allied to the vaso-motor disturbances. An excessive development of the panniculus adiposus sometimes forms upon the affected side, and even the bones may undergo hyperplasia. In other cases atrophic processes are observed, likewise herpes, acne, lichen, and erysipelas on the affected parts. The hairs may become rough, fractured, suddenly grow gray or fall out. In some cases we notice an alternation of pigmented with white (unpigmented) hairs, the latter having developed during the individual attacks of neuralgia. Among the rarer phenomena are ophthalmia neuroparalytica, which has been attributed to functional disturbances of certain trophic fibres in the trigeminus. Glaucoma and, according to Bull, iritis and chorioiditis have also been observed after trigeminal neuralgia.

The sensibility of the integument of the affected nerves is not infrequently changed. It is generally heightened at the beginning of the disease, later it is diminished. Sensory disturbances are sometimes noticeable only during the attacks.

Involuntary contractions of the facial muscles are sometimes observed during the seizures, so that tic douloureux is complicated with tic convulsif. In certain cases contractions occur in the muscles of the neck and limbs; in Sinklar Holden's case there were widespread tonic muscular spasms.

The duration of the disease may vary from a few days to a number

of years; it sometimes lasts a lifetime. It disappears occasionally during intercurrent diseases, or alternates with neuralgia of other nerves. Relapses are frequent, and sometimes recur after the lapse of years.

Some patients suffer from profound melancholy, and may even be driven to commit suicide. Others become shy, because noisy surroundings provoke an attack. Still others lose strength from loss of sleep or refusal to take food (because mastication gives rise to a paroxysm).

IV. DIAGNOSIS.—The diagnosis is easy, if we bear in mind the paroxysmal character of the pain, its distribution, and the presence of painful points.

1. *Ophthalmic neuralgia.* The pain is situated in the upper lid, the frontal region as far as the vertex, the eyeball and its socket, the base of the nose and its integument as far as the tip, the anterior portion of the nasal cavity (vide Fig. 27).

FIG. 27.
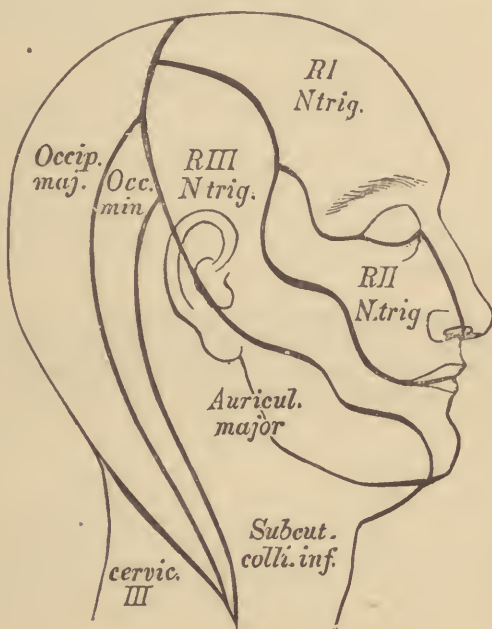
Distribution of the trigeminus to the face.

a. Supraorbital neuralgia : pain in frontal region, upper lid, and root of nose. Pressure points ; the most constant one at the supraorbital foramen or notch, less constantly on the upper lid (palpebral point), the parietal eminence (parietal point), the inner angle of the eye or cartilage of the nose; sometimes the entire nerve tract is sensitive.

b. Ciliary neuralgia. Most violent pain in the eyeball; a symptom of many ocular diseases.

2. *Supramaxillary neuralgia.* Pain situated in lower lid, cheek, upper lip, lateral part of nose, region of malar bone, and anterior part of forehead, upper row of teeth, gums, and nasal cavity.

a. Infraorbital neuralgia. Situation of pain: lower lid, cheek, upper lip, lateral part of nose; sometimes upper teeth, and mucous membrane of cheek; if the nervus subcutaneus malæ is involved, in region of malar bone, and anterior part of forehead. Pressure points : most constantly at infraorbital foramen, more rarely on the upper lip (labial point), malar region (malar point), alveolar processes of upper jaw (alveolar point), rarely on the gum.

4

b. Alveolar neuralgia is confined to the alveolar processes of the upper jaw.

3. *Inframaxillary neuralgia.* Situation of pain: region of chin and lower jaw, mucous membrane of cheek, inferior alveolar process, tongue, external ear, and forehead.

a. Mental neuralgia. Pain in the chin; pressure point at the mental foramen.

b. Lingual neuralgia (glossalgia). Pain in one-half of the tongue; sometimes unilateral coating and increased thickness of the tongue. Painful point at the side of the tongue; sometimes increased salivation, and involuntary movements of the tongue.

c. Inferior alveolar neuralgia. Pain in lower alveolar processes.

d. Auriculo-temporal neuralgia (very rare). Pain in external auditory canal, concha, and temple.

V. PROGNOSIS.—This depends in great part upon the etiological factors. Not all cases in which no cause can be ascertained present a favorable prognosis. If heredity plays a part in the etiology, we must expect great obstinacy and frequent relapses of the disease. The longer the neuralgia has lasted, the slighter are the chances of recovery. The prognosis of intermittent neuralgia is favorable.

VI. TREATMENT.—The causal treatment varies, of course, with the etiology. In intermittent neuralgia we may give quinine (gr. xv., two hours before the expected attack, for three days in succession); if quinine is not tolerated, it is replaced by Fowler's solution (ten drops t. i. d., after meals). If the disease is the result of exposure, we should order diaphoretics, give salicylic acid internally (gr. vij. every hour, until tinnitus aurium is produced), and cover the parts with wadding. Syphilitic cases are treated with iodine and mercury, anæmic cases with iron, etc.

In the symptomatic treatment of trigeminal neuralgia we regard quinine and arsenic as the most important internal remedies. Quinine often does not act until given in very large doses (gr. lxxv. or more).

Among other internal remedies may be mentioned: Aconit. (℞ Aconitin., gr. ¾; ext. hyoscyam., gr. xv.; pulv. liq., q. s. u. ft. pil. No. 50. D. S. One to two pills morning and evening); Colchicum (℞ Tinct. colchici, ʒ iij.; tinct. aconiti, ʒ iss. M. D. S. Fifteen drops t. i. d.); Acid salicylic., gr. xv. every hour; potassium iodide, gr. x. t. i. d.; ℞ Argent. nitrat., gr. xv., argill. albæ, q. s., f. cum aq. dest. q. s. pil. No. 100. D. S. One pill t. i. d., after meals; ℞ Auro-natrium chloratum, gr. v.; ext. dulcamar., gr. l., f. pil. No. 50. D. S. One pill t. i. d; phosphorus, zinc, mercurial and copper preparations; opium, belladonna, strychnia, chloral hydrate, butylchloral (℞ Butylchloral hydrat., gr. lxxv.; glycerin., ʒ v., aquæ, ʒ iv. M. D. S. One tablespoonful every five to ten minutes), potassium bromide (ʒ i.–iij. in a single dose), tinct. gelsemii (five to twenty drops t. i. d.), amyl nitrite (five drops by inhalation until the face is reddened), etc.

Among external remedies the most important is electricity, then follows the subcutaneous injection of narcotics.

As a rule, the galvanic current is indicated. Feeble currents should be employed; its strength may be gradually increased during the sitting; the stabile application is preferable; the anode is placed upon the affected parts, especially upon pressure points; the cathode, to the back of the neck or indifferent part. The sitting should not last longer than three to five minutes, but sometimes two to three sittings daily are advisable. Some authors recommend descending currents, *i. e.*, the anode central, the cathode on the peripheral parts. In intra-cranial disease, the current is passed transversely through the skull.

The faradic current may be employed with the wire-brush, but this can rarely be applied to the face, on account of the severe pain produced thereby. M. Meyer showed that it sometimes acts very well when applied to the back of the neck.

The remedies which may be employed subcutaneously are morphine (gr. iss. : ℥ i., 1 syringeful), atropine (gr. ss. : ℥ i., ¼–½ syringeful), strychnine (gr. iss. : ℥ i., ¼–½ syringeful), chloroform, and ether. The patients themselves should not be allowed to make injections of morphine, since they are apt to fall into the morphine habit. It is a noteworthy fact that patients suffering from neuralgia often present a remarkable tolerance to narcotics. For example, Trousseau recommended as much as ℨ iiss. of opium or ℨ i. of morphine daily.

Among other external methods of treatment may be mentioned : aquapuncture, inunctions of veratrine, belladonna, morphine, chloroform, ether, croton-oil collodion—linear cauterization along the nerves—blisters, leeches, chloroform poured on cotton and placed behind the ear, and compression of the nerves.

In desperate cases, surgical operations may be resorted to, such as ligature or compression of the carotid, nerve stretching, neurotomy, and neurectomy.

Compression of the carotid was successfully employed by Earle, Gerhardt, and Seifert. Nussbaum and Patruban have had good results from ligature of the artery. Nerve stretching is the mildest operation, but the most uncertain in its effects. Nor can neurotomy be regarded as a radical operation. Neurectomy offers the greatest chances of success, but relief can only be expected with certainty if the operation has been performed on the central side of the site of the neuralgia. Experience has shown, however, that the neuralgia is sometimes relieved for a longer or shorter period, even if the affected part was not removed.

A. Wagner collated 134 cases of excision of the nerve in trigeminal neuralgia, and found:

Recovery for years	25	times	(18.7%).
" " months	18	"	(13.4%).
Operation unsuccessful	9	"	(6.6%).
Death.	6	"	(4.5%).
Result unknown	24	"	(18.0%).
Relapses	52	"	(38.8%).

2. Cervico-Occipital Neuralgia.

(Occipital Neuralgia.)

I. ETIOLOGY.—Cervico-occipital neuralgia affects branches of the cervical plexus (four upper cervical nerves). The occipitalis major is most frequently affected, in rarer cases the other sensory branches, viz., occipitalis minor, auricularis magnus, subcutaneus colli inferior, and supraclavicular.

Under unfavorable circumstances, accordingly, the pain may extend over the back of the neck and occiput, the posterior surface of the ear, the anterior and lateral parts of the neck, scapula, and upper part of the chest.

The disease is more frequent in women, from the 20th to 50th years of life. Heredity is rarely demonstrable; the affection is often found in anæmic, hysterical, and nervous individuals.

Exposure is mentioned as the cause of many cases; in others it is the result of injury. Compression of the nerves by swollen lymphatic glands, tumors, or dilatation of the vertebral arteries is found occasionally. In some patients, the disease is the result of affections of the vertebræ or cervical cord (tuberculosis, osteoma, gumma, periostitis of the vertebræ, meningitis, hemorrhage, etc.). In a few cases it is associated, perhaps, with catarrhal disease of the middle ear. Several cases have been observed after infectious diseases, or as a reflex symptom of intestinal affections, perhaps also of the eruption of the wisdom teeth. Intermittent forms dependent on malarial infection are much rarer than the corresponding forms of trigeminal neuralgia. It is sometimes radiated from trigeminal neuralgia. Obstinate cases occur with relative frequency in uræmia.

II. SYMPTOMS.—The symptoms of pure occipital neuralgia consist of paroxysms of pain which start in the upper part of the back of the neck, and radiate along the occiput to the vertex (vide Fig. 27). The pains are sometimes excruciating, and a dull sensation of pain and peculiar feeling of stiffness in the neck often persist during the inter-paroxysmal period. The movements of the head are often interfered with.

The duration and number of the paroxysms are subject to great variations. The attacks sometimes occur spontaneously, sometimes follow excitement, vomiting, coughing, careless rotation of the head, pressure on the back of the neck, etc. Both nerves are not infrequently affected, one more markedly than the other. The pains sometimes radiate into remote nerve tracts. This occurs with relative frequency into the trigeminus (anastomosis with the frontal nerve) in the temples, more rarely into a larger part of the trigeminal distribution, or into the brachial plexus.

The most constant pressure points are the occipital point (between the mastoid process and first cervical vertebra, corresponding to the exit of the occipital nerve) and the parietal point (upon the parietal eminence). More remote painful points may be found on the spinous processes of the cervical vertebræ. Slight contact with the painful points often produces neuralgic attacks. The entire course of the nerve is often tender on pressure.

The affected parts are almost always hyperæsthetic at the beginning of the disease.

Vaso-motor disturbances are often present, and are probably produced by sympathetic nerve fibres, since they are not infrequently associated with pupillary changes. These symptoms consist of redness of one-half the face or the ear, injection of the conjunctiva, sometimes lachrymal and nasal hypersecretion, unusual warmth and feeling of increased heat in the face; the pupils are generally narrow, more rarely dilated. Some patients complain of difficulty of hearing and ringing in the ears.

Trophic changes have been rarely described. Rosenthal noticed alopecia, Romberg observed small swellings, which developed upon the occiput during the attack and disappeared after its cessation.

Rheumatic neuralgia is attended not infrequently with swelling of the cervical lymphatic glands.

During violent seizures, tonic or clonic muscular spasms may develop in the face, limbs, or throughout the entire body. Violent vomiting during the attacks has also been described.

The duration of the disease varies from a few weeks to several months or years. Incurable cases are much rarer than in trigeminal neuralgia.

Neuralgias of the other nerves belonging to the cervical plexus are readily recognized by the following symptoms:

Neuralgia of the occipitalis minor gives rise to pain in the lateral part of the occiput as far as the ear; painful point behind the mastoid process at the exit of the nerve beneath the skin (vide Fig. 27).

b. *Neuralgia of the auricularis magnus* causes pain on the posterior aspect of the concha, above the mastoid process and over the parotid; painful point a little above the middle of the neck, between the trapezius and sternomastoid, and also upon the concha.

c. *Neuralgia of the subcutaneus colli inferior :* pain in the lower, middle, and anterior parts of the neck; painful point, same as in neuralgia of auricularis.

d. *Neuralgia of the supraclavicular :* pain in the acromial region, shoulder, and upper part of the chest.

III. DIAGNOSIS.—The diagnosis is easy, but it must not be forgotten that these neuralgias sometimes precede for years a latent affection of the cervical vertebræ and spinal cord. Hence these parts should be carefully examined in obstinate cases.

IV. PROGNOSIS.—This is much more favorable, as a general thing, than that of trigeminal neuralgia, but depends chiefly on the etiology.

V. TREATMENT.—The treatment, in general, must follow the principles laid down in the consideration of trigeminal neuralgia. The chief remedies are quinine internally, morphine subcutaneously, and the galvanic (more rarely the faradic) current transversely through the upper part of the nucha. In recent cases we may employ flying blisters and warm applications. In obstinate cases surgical interference may be resorted to.

3. *Phrenic Neuralgia.*

1. The occurrence of neuralgia of the phrenic nerve has been reported a number of times by the older authors, and more recently by French writers.

2. The disease is said to occur either as an independent affection or during the course of diseases of the pleura, pericardium, myocardium, aorta, liver, spleen, kidneys, stomach, intestines, and peritoneum. Under such circumstances it may take a prominent part in the symptomatology, and, in angina pectoris, for example, may be the cause of the overpowering pains. In addition, exposure, injury, hysteria, and epilepsy have been mentioned as causes.

3. The chief symptom is pain, located chiefly at the base of the chest, but occasionally running along the entire course of the nerve. The pains increase paroxysmally, but not infrequently persist in a less degree; they may be unilateral or bilateral. The pain often radiates into remote nerve tracts—into the arms, neck, chin, nucha, but most constantly into the shoulders.

Pressure points are found in the following localities : a. anterior points of insertion of the diaphragm, especially on the ninth rib; b. posterior points of insertion of the diaphragm, especially the lowermost rib; c. lateral part of the neck upon the scalenus anticus; d. on the sternum at the level of the second and third intercostal spaces; e. over the spinous processes of the second and fifth, rarely the sixth cervical vertebræ.

We usually find disturbances of the respiratory movements: jerky, superficial, painful inspiration, singultus, yawning, etc. Deglutition may also be interfered with. Great terror and a feeling of impending dissolution are sometimes present. Relapses may occur.

4. The diagnosis is difficult. The disease cannot always be distinguished from an affection of the diaphragmatic pleura or the peritoneum.

5. Treatment: Mustard poultices, leeches, cups, blisters, injections of morphine, electricity.

4. *Cervico-Brachial Neuralgia.*

I. ETIOLOGY.—The brachial plexus is derived from the four lower cervical and first dorsal nerves. Neuralgias of this plexus are not rare, and occur somewhat more frequently in men than in women.

Some cases are the result of exposure, a much larger number are due to injury.

The traumatic causes include fall, blow, incised or gunshot wounds, fracture or dislocation, formation of callus, aneurism of the aorta or subclavian artery, swollen glands in the axilla, neuromata or other tumors, tuberculosis or cancer of the spine, phlebectomy, wounds, injuries to the little fingers, amputation-neuromata. Allied to this form is that variety of neuralgia produced by over-exertion of the muscles, as in playing piano, sewing, etc.

Brachial neuralgia sometimes follows articular affections of the elbow, hand, or shoulder.

It is sometimes radiated from trigeminal or cervico-occipital neuralgia, angina pectoris, diseases of the liver and spleen. According to Salter, it may be a reflex effect of dental irritation.

Chlorosis, anæmia, hysteria, malaria, and lead poisoning appear to be the starting-point of the disease in some cases.

II. SYMPTOMS.—The disease is almost always unilateral. Bilateral neuralgia has been reported as the result of cancer of the spine and of over-exertion in gymnastic exercises. The right arm seems to be affected more frequently than the left.

As a rule, a number of the cutaneous nerves are affected simultaneously, and motor disturbances are generally present.

The chief symptom is pain, which may be shooting, boring, burning, etc. Pains of unusual intensity have been observed after gunshot wounds of the nerves (causalgia). The patients are often unable to localize the pains. They sometimes radiate into the neck, occiput, face, or intercostal spaces. In many places the pains are constant, but present paroxysmal exacerbations. The paroxysms are especially apt to occur at night after going to bed, or after a careless movement of the arm. Some patients experience relief from carrying the arm in a sling or supporting it with the other hand; others are relieved by extension of the limb.

The painful points vary according to the affected nerve tracts; they are not constant, and sometimes entirely absent. In radial neuralgia, the pressure points are: The point of flexion of the nerve in the arm, and the dorsal surface of the forearm immediately above the wrist. In ulnar neuralgia: A point between the internal condyle and olecranon, and the anterior surface of the forearm immediately below the head of the ulna. In median neuralgia, the painful points are: The bicipital sulcus, the fold of the elbow, the radial side of the volar surface of the forearm immediately over the head of the radius. Other pressure points are: The supraclavicular and axillary fossæ, lower angle of scapula, and the points of exit of the various cutaneous nerves.

The more remote pressure points are the spinous processes of the four lower cervical and two or three upper dorsal vertebræ.

Circulatory changes (pallor or redness of the skin) are not infrequent. There is sometimes an abnormally profuse formation of sweat.

Among the trophic changes may be mentioned herpes zoster, urticaria, eczema, pemphigus, increased growth of hair, changes in the nails, glossy skin, etc.; the muscles often undergo atrophy.

Motor disturbances are frequent, and consist of fibrillary twitchings, a feeling of stiffness, paresis, paralysis, more rarely tonic and clonic spasms. The patients often complain of a feeling of rigidity and stiffness in the muscles, and the fingers are permanently extended like claws.

Gross muscular power may be better retained than fine manipulations, such as writing, sewing, etc.

Paræsthesiæ (feeling of coldness, formication) are frequently mentioned.

III. DIAGNOSIS.—The diagnosis of brachial neuralgia is easy, but the recognition of the etiology and site of the disease may be very difficult or even impossible (vide Figs. 10 and 11).

IV. PROGNOSIS.—This depends almost entirely upon the etiology. The neuralgia generally disappears if the cause is removed.

V. TREATMENT.—The treatment follows the same general principles that have been laid down in the consideration of trigeminal neuralgia. The local measures include: carrying the arm in a sling, absolute rest, subcutaneous injections of morphine, and electricity. The galvanic current is generally employed; stabile application, anode upon the painful points or the affected nerve trunks, or a descending current along the nerves.

Nerve stretching is said to have been successful in some cases. Neurectomy should not be performed, as a rule, because it gives rise to paralysis.

5. *Dorso-intercostal Neuralgia.*

(*Intercostal Neuralgia.*)

I. ETIOLOGY.—The intercostal nerves, after leaving the intervertebral foramina, divide into a posterior (dorsal) and anterior (intercostal) branch. Neuralgia occurs most frequently in the latter, rarely in the dorsal branches alone, somewhat more frequently in both.

The site of the pain is very extensive, since the dorsal branches supply the integument of the back down to the crest of the ilium, the intercostal branches supply the lateral and anterior surfaces of the chest and abdomen as far as the symphysis pubis. The first dorsal nerve also passes into the brachial plexus, and supplies the inner surface of the arm.

Intercostal neuralgia is more frequent in women than in men, and from the age of twenty to fifty years. Anæmia, chlorosis, and nervousness possess a still greater etiological significance than in many other neuralgias.

Exposure and traumatism are mentioned as frequent causes.

The traumata include: a fall, blow, fracture of the ribs and unfavorable development of callus, tuberculosis of the ribs, neuromata and other tumors, pressure of aneurisms on the spine; tuberculosis, cancer, periostitis, syphilis of the vertebræ or spinal meninges, etc.

Intercostal neuralgia is sometimes the result of diseases of the respiratory organs, heart, stomach, intestines, liver, or spleen.

It is associated not infrequently with pleurisy and pulmonary phthisis, and also with nervous affections of the heart; on the other hand, cardiac neuroses are apt to develop after an attack of neuralgia. Intercostal neuralgia has also been known to develop after gastralgia, or painful affections of the liver and spleen.

It sometimes develops during convalescence from severe diseases, particularly typhoid fever. Malaria is a not infrequent cause.

Intercostal neuralgia may also appear as one of the effects of lead

poisoning, as a reflex symptom of uterine or ovarian disease, and as a radiated symptom in trigeminal, occipital, and cervico-brachial neuralgia.

II. ANATOMICAL CHANGES.—Very little is known in this regard. Thickening and hyperæmia of the neurilemma, degeneration of the nerve fibres, and neuromata have been observed in a few cases. The disease is not infrequently a true neurosis, which does not present any anatomical changes.

III. SYMPTOMS.—Intercostal neuralgia is commonly unilateral; cases of bilateral disease are much rarer. It occurs more frequently on the left side. This is attributed to the fact that the venous blood on the left side passes in a roundabout way through the vena hemiazygos to the inferior vena cava, so that stasis and compression of adjacent structures are more likely to happen on this side. As a rule, two or three adjacent intercostal nerves are affected at the same time. The fifth to ninth intercostal nerves are involved most frequently.

The pain may occur in paroxysms, or may persist constantly in a milder degree. It may be so violent and obstinate as to deprive the patient of sleep. The pain is described as burning, sticking, lancinating, etc. It sometimes extends half-way around the thorax, sometimes is especially violent in circumscribed localities. It often radiates into the arm. If the disease is bilateral, the patients experience the sensation of a constricting band around the thorax. Coughing, sneezing, deep respirations, loud talking, etc., produce paroxysms of pain or intensify existing pains. On this account the patients often speak in a low tone, breathe superficially, and sometimes look like asthmatic individuals. Slight pressure on the skin may intensify the pain; firm pressure not infrequently relieves it. The patients sometimes assume a bent position, the spine usually presenting a convexity towards the healthy side.

There are three principal painful points: one immediately adjacent to the spine, corresponding to the exit of the nerve from the intervertebral foramina (vertebral point), the other at the middle of the nerve where the nervus perforans lateralis ramifies in the lateral part of the thorax (lateral point), and a third along the edge of the sternum (sternal point) or on the rectus abdominis, where the anterior perforating nerve makes its exit. Pressure points are also found not infrequently on the spinous processes of the dorsal vertebræ.

Cutaneous hyperæsthesia is often found in the distribution of the affected nerve, sometimes diffusely, sometimes in circumscribed spots; anæsthesia is observed more rarely, and usually in old cases.

Trophic disturbances are often present, herpes zoster being the most frequent. The neuralgia may precede or follow the zoster. If zoster occurs without neuralgia (generally in children), it must be assumed that only the trophic fibres are diseased, and that the sensory fibres have escaped.

Woaker noticed foul-smelling perspiration during the attacks.

The course and duration of the disease depend on the causes. In a number of cases it is followed by hysteria or cardiac neuroses.

IV. DIAGNOSIS.—The recognition of the disease is easy. In rheumatism of the muscles of the chest, the muscles themselves are sensitive to pressure, and the pain is constant. In pleurisy, other changes in the respiratory apparatus are present. Inflammation of the ribs gives rise to a visible swelling. In making a diagnosis, we should not be satisfied until the etiology is established.

V. Prognosis is unfavorable only when the disease is produced by incurable lesions.

VI. Treatment.—The treatment is causal and local. The latter as in other neuralgias : morphine subcutaneously, and the galvanic, more rarely the faradic, current. The current should be strong, stabile, the anode to the painful points, or a descending current along the affected nerves. In other respects the treatment is similar to that of trigeminal neuralgia. Nussbaum recently performed nerve stretching, but the results were not permanent.

Appendix.

Neuralgia of the Mammary Gland (Mastodynia) is a variety of intercostal neuralgia, since the breast is supplied by the second to sixth intercostal nerves, in addition to the supraclavicular nerves. Neuralgic affections of the breast occur almost exclusively in women. It hardly ever develops before puberty, usually from the sixteenth to the thirtieth year, rarely later, Anæmia, chlorosis, hysteria, and nervousness not infrequently form the basis of the disease. In some cases it is attributed to injury, uterine affections, protracted lactation, and menstrual disturbances. Nodular indurations are sometimes found in the gland, and often disappear with the cessation of the neuralgia.

Mastodynia is often bilateral; according to some writers it is more frequent on the left side. The patients complain of a violent burning or lancinating pain which increases paroxysmally and sometimes lasts several hours. Vomiting may occur at the height of the paroxysms. The slightest contact with the skin increases the pains, and their intensity is also increased, as a rule, shortly before menstruation (congestion of the breast?). The patients often experience a sensation of tension and weight in the nipple. The pain is either diffused over the entire gland or it is localized in certain points. It often radiates into the neck, shoulders, arms, and back.

Pressure points are not constant or characteristic. The nipple is generally very sensitive to pressure, often also circumscribed parts of the gland. Painful points are also found on the spinous processes of the lower cervical and upper dorsal (especially the second and fifth) vertebræ.

A secretion of milk or a colostrum-like fluid has been observed in a few cases after each paroxysm of pain. Alfter observed herpes zoster after mastodynia.

The disease may last many years and drive the patient to despair.

The diagnosis is usually easy. Inflammatory conditions are associated with cutaneous changes and elevated temperature : malignant growths, which may produce similar pains, present a constant growth.

The prognosis is not always favorable.

Treatment.—The breast should be kept elevated. Inunction with narcotic ointments. Cooper recommended :

R Extract. Belladonnæ,
    Cerat. Cetacei............... ....āā ℥ ss.
M. D. S. Externally.

Subcutaneous injections of morphine, electricity. Due attention should be paid to anæmia or other predisposing conditions. As a last resort the breast may be extirpated.

6. *Lumbo-Abdominal Neuralgia.*

Lumbo-abdominal neuralgia affects the short nerves of the crural plexus—iliohypogastric, ilio-inguinal, lumbo-inguinal, and external spermatic. *a.* The iliohypogastric nerve is distributed to : the integument of the hip and upper part of the hypogastrium. *b.* Ilio-inguinal nerve : integument of the mons veneris and tensor fasciæ latæ muscle. *c.* Lumbo-inguinal nerve : median part of the groin and thigh half-way down. *d.* External spermatic nerve : scrotum or labium majus and inner surface of the thigh.

As a rule, several or all of these nerves are affected at the same time. The pain is generally felt in the integument of the loins as far as the buttocks, the hypogastrium, mons veneris, scrotum (or labium majus) and the inguinal region.

Among the causes mentioned are: exposure, diseases of the spine and meninges,

pelvic exudations or tumors, and flexions of the uterus. The neuralgia is more frequent on the left side.

The pains possess the ordinary neuralgic character. Pressure points are found along the spine (lumbar point), in the middle of the crest of the ilium (iliac point), on the scrotum, labium, occasionally on the cervix uteri. The neuralgia may be associated with contraction of the cremaster muscle, priapism, discharge of semen, leucorrhœa, and vesical tenesmus.

The treatment is the same as in other neuralgias.

7. *Crural Neuralgia.*

1. Crural neuralgia is a rare affection. It is observed most frequently among the laboring classes in males, in whom exposure and over-exertion are not infrequent causes. It may also be the result of injury : compression by exudations around the spine, psoas and pelvic organs, pressure of the uterus or swollen lymphatic glands in the pelvis and groins, aneurism of the crural artery, inguinal hernia, luxation of the femur, stab and gunshot wounds, etc. Seeligmueller claims that sprain of the ankle joint often gives rise to neuralgia of the saphenus major nerve. Crural neuralgia sometimes follows sciatica.

2. The pains extend along the inner surface of the thigh to the knee, and following the course of the saphenus major nerve, may pass along the inner surface of the leg to the great toe. The neuralgia is sometimes confined to a single cutaneous branch. The pains are increased on movement of the limb, and are often more severe at night. They occasionally radiate into more remote regions, particularly into the loins. The pressure points are: *a*. Crural point, below Poupart's ligament, corresponding to the exit of the crural nerve; *b*. Anterior thigh point, at the exit of the saphenus minor through the fascia lata; *c* Knee point, inner surface of the knee joint; *d*. Plantar point, immediately in front of the ankle; *e*. Toe point, at the base of the big toe.

Hyperæsthesia is often, anæsthesia is rarely present. Some patients complain of formication, coldness, and a feeling of stiffness. Vaso-motor or trophic disturbances are rare.

3. The treatment, apart from causal treatment, is the same as that of sciatica.

8. *Obturator Neuralgia.*

Obturator neuralgia possesses more of a surgical interest, because it is an important sign of incarceration of herniæ in the obturator foramen. Pains then develop upon the inner surface of the thigh as far as the knee, associated with numbness, formication, and usually with inability to adduct the thigh (because the motor fibres of the nerve have also been compressed). The treatment consists in reposition of the hernia.

9. *Neuralgia of the External Cutaneous Nerve.*

The pains extend along the outer surface of the thigh as far as the knee. The disease is generally associated with crural neuralgia, and rarely exists alone.

10. *Sciatica.*

(*Ischialgia.   Malum Cotunnii.*)

I. Etiology.—Neuralgias of the sciatic nerve are very common. They are more frequent in men than in women, exceptional in childhood, and develop, as a rule, between the ages of twenty and sixty years.

Anæmia, chlorosis, and hysteria possess much less etiological importance than in neuralgias of other nerves.

Exposure to cold and wet, and injury, on the other hand, are the most frequent causes of the disease.

Traumata affecting the sciatic nerve include: tumors and inflammations of

the meninges, with compression of the sciatic nerve; tuberculosis, cancer, gumma, exostoses, and periostitis of the vertebræ, curvature of the spine, pelvic inflammations and tumors, flexions of the uterus, compression by the pregnant uterus, difficult labor and compression by the child's head, distention of the rectum with fæces or foreign bodies, dislocation or fracture of the femur, exostoses on the bones of the lower limbs, sciatic hernia, fall or blow on the buttocks, constant sitting and riding, heavy lifting; incised or gunshot wounds; neuromata and other tumors with pressure on the nerve; aneurism of the abdominal aorta and popliteal artery, etc.

Sciatica is sometimes associated with constitutional diseases, for example, gout. It sometimes precedes diabetes mellitus and locomotor ataxia, probably as the result of central causes.

Certain cases are the result of infectious diseases, viz.: malaria, syphilis (without the formation of gummata), gonorrhœa, typhoid fever (particularly during convalescence).

In rare instances it is the result of toxic influences, for example, in lead and mercury poisoning.

Whether hemorrhoids and checked sweating of the feet give rise to sciatica has not been determined with certainty.

Climatic and meteorological conditions are not devoid of influence in this disease. Some English and German writers report an unusual frequency of the disease in certain localities. It is known to every experienced physician that cases of the disease are more frequently observed during cold seasons in which there are rapid changes of temperature.

II. ANATOMICAL CHANGES.—These may be entirely absent, even if the disease has lasted a long time. In other cases hyperæmia of the nerve, varicose dilatation of its blood-vessels, swelling and increase of the connective tissue, atrophy and fatty degeneration of the nerve fibres have been observed. Josset recently reported a case in which an obstinate sciatica was cured by puncture of the nerve sheath and evacuation of half an ounce of serum.

III. SYMPTOMS.—As a rule, sciatica is unilateral, though Hasse maintained that it is not so infrequently bilateral as is generally believed, but that the pain on one side may be comparatively slight, and therefore overlooked. The disease sometimes begins on one side and then passes to the other, sometimes it is bilateral from the start, particularly in diseases of the meninges or spine.

The pain may affect the entire distribution of the nerve, or be confined to individual branches (generally the posterior cutaneous, occasionally the plantar nerve). In the former event the pain extends over the buttock, posterior surface of the thigh, the entire leg and foot, with the exception of their inner surface (vide Figs. 22 and 23).

As a rule, the pains are constant, but their intensity increases in paroxysms, which may assume an intermittent character, even when produced by organic lesions. They are described as burning, tearing, sticking, boring, etc. As a rule they run from above downwards, rarely in the opposite direction. The paroxysms often develop spontaneously, in other cases they are produced by an incautious movement of the limb, slight pressure, laughing, sneezing, etc. In one of my patients unusually violent paroxysms occurred at the period of menstruation. As a rule, the paroxysms subside so much more quickly the more quiet the patient keeps. Some patients, on the other hand, are relieved while walking about, or firmly compressing the sciatic nerve.

Vomiting sometimes occurs at the height of the paroxysm. Tonic or clonic twitchings are noticed occasionally, and the heel may be drawn

against the buttock. The pains radiate not infrequently into the sacral region or the distribution of the crural nerve. Sciatica may also be associated with intercostal neuralgia.

Pressure points may be entirely absent, in other cases they are inconstant. The nerve is sometimes tender on pressure along its entire course.

Among the most frequent painful points are : *a*, a point along the sacrum at the level of the posterior superior spine of the ilium; *b*, lower border of gluteus maximus at the exit of the nerve from the sciatic foramen; *c*, immediately behind the trochanter major; *d*, middle of the posterior surface of the thigh, corresponding to the exit of the posterior cutaneous nerve; *e*, bend of the knee (tibial nerve); *f*, immediately below the head of the fibula (peroneal nerve); *g*, behind the malleoli; *h*, several points on dorsum of foot or the calf. The sacral plexus is sometimes found to be tender on pressure when examined per vaginam or rectum.

Changes in the color of the skin and trophic disturbances are generally absent. In very rare cases mention is made of abnormal redness of the skin, increased heat and diaphoresis, increased growth of hair, outbreak of herpes zoster and furuncles.

Sensibility may be intact, but hyperæsthesia or anæsthesia has been observed in a number of instances. The patients complain not infrequently of paræsthesiæ: a feeling of coldness, pricking, burning, formication, stiffness.

Emaciation of the muscles with secondary paretic symptoms is not infrequent in chronic sciatica, and is often the result of disuse of the limb. But marked atrophy sometimes develops very early and rapidly, and has been attributed by Landouzy to neuritic changes in the sciatic.

In one case Graves observed hypertrophy of the muscles.

The patellar tendon reflex was unchanged in all of my cases.

The patients often assume a peculiar position. They generally lie upon the healthy side, with the thigh drawn up and the knee bent, in order to avoid stretching the nerve. They avoid walking altogether or tread upon the floor very carefully, favoring the limb as much as possible. Many patients complain of violent pain while sitting, and are compelled to assume a recumbent position almost constantly.

Increased bodily temperature is sometimes observed at the beginning of the disease. The patients often complain of constipation, associated with increasing violence of the paroxysms of pain. Braun showed that the urine not infrequently contains sugar.

Sciatica may run its course in two to six weeks, but is more often chronic. Cases have been known to last more than thirty years. In rare cases spontaneous recovery ensues at the end of years. There is always a great tendency to relapses.

IV. DIAGNOSIS.—The diagnosis is easy, inasmuch as the pains often run along the exact anatomical course of the disease.

It may be mistaken for: *a*, coxitis, which is excluded by the characteristic position of the body, pain on rotation of the limb or on compression against the acetabulum; *b*, psoitis, in which a characteristic position is also noticeable; *c*, muscular rheumatism, the pain is irregularly distributed, and is produced particularly by pressure on the muscles; *d*, hysterical joint affections, in which a diagnosis can often be made only after prolonged observation.

V. PROGNOSIS.—This varies according to the etiology of the disease.

VI. TREATMENT.—Apart from causal treatment, the leg should be

kept quiet, the food should be easily digestible, and daily evacuation from the bowels secured. The entire limb should be rubbed every morning and evening with Stokes' liniment:

℞ Ol. terebinthin.,
  Aq. communis..............................āā ℥ xiv.
  Vitelli ovi...................................i.
  Ol. lini...................................℥ iss.
F. linimentum.

and the limb then wrapped in cotton batting; iodide of potassium (℥ iij. : ℥ vij., one tablespoonful t. i. d.) should be given internally. This treatment will suffice in many cases.

I have seen good and permanent results in a number of cases from change of air.

Electricity justly plays a prominent part in treatment, although I have seen its application followed by an aggravation of the symptoms in a number of fresh cases.

The galvanic current is usually preferred: large electrodes, a strong current, anode stabile to the painful points, or a descending stabile current, or successive galvanization of small, consecutive stretches of the nerve. Benedikt introduced one pole into the rectum, and placed the other upon the sacrum. Ciniselli recommended the constant application of zinc and copper plates, connected by a metallic wire. Some authors recommend the use of the faradic current, either as the electric brush or moxa.

Great benefit may be derived from baths of all kinds.

Among other methods of treatment may be mentioned: *a.* Derivatives: cups, blisters, moxa, actual cautery, aquapuncture, subcutaneous injection of nitrate of silver, alcoholic inunctions, inunctions with veratrine or croton oil, etc. *b.* Narcotics: morphine or atropine subcutaneously, belladonna in ointment or endermically, chloroform and chloral hydrate by enema, etc. *c.* Antirheumatics: aconite, colchicum, potassium iodide, salicylic acid, etc. *d.* Specifics: turpentine, arsenic, quinine. Tinct. gelsemii, 5–20 drops t. i. d., had a very rapid effect in several of my cases. Neuber recently recommended osmic acid subcutaneously in all kinds of neuralgia. In my own hands, this remedy (gr. viij. : ℥ iij., a half-syringeful subcutaneously) gave no results in a large number of cases, and many patients complained of severe, sometimes very protracted pain at the site of injection.

Among the surgical remedies may be mentioned massage and nerve stretching. Nerve stretching has recently been performed as a bloodless operation, the limb being flexed as strongly as possible upon the abdomen. In one case I obtained temporary good effects from this plan.

11. *Spermatic Neuralgia.*

In spermatic neuralgia, attacks of pain occur in the testicles and epididymides, and extend along the seminal duct to the loins. The testicles and epididymides are tender on pressure, and are sometimes swollen. The pain may be so violent that the patient is covered with cold sweat, and suffers from chattering of the teeth, attacks of syncope, convulsions, and vomiting. The left testicle is most frequently affected.

The disease often develops at the period of puberty. The patients are often pale, nervous individuals, who have practised masturbation or indulged in sexual excesses. In other cases, the disease has been attributed to sexual continence. Injury, exposure, and varicocele are also regarded as causes. Inter-

mittent neuralgia, which disappears after the administration of quinine, is sometimes observed.

The majority of German writers believe that the disease is an affection of sympathetic fibres in the testicles.

The treatment consists of elevation of the testicle by means of a suspensory, inunctions with belladonna ointment, subcutaneous injections of morphine, lukewarm baths, electricity, iron, quinine, arsenic, or potassium bromide. Castration has been performed in obstinate cases.

APPENDIX.

A number of other neuralgic affections, which cannot easily be localized, may develop in the external genitals and perineal region. They are usually the result of cold, injury, onanism, and sexual excesses. We may briefly mention:

a. Neuralgia of the penis and glans penis: attacks of pain in the penis or glans, sometimes associated with priapism, involuntary discharge of semen, and disturbances of micturition.

b. Neuralgia of the scrotum (or labia majora).

c. Neuralgia of the urethra.

d. Ano-vesical neuralgia: spasm of the sphincters of the rectum and bladder, with hyperæsthesia (more rarely anæsthesia) of the perineum.

e. Ano-perineal neuralgia.

12. *Coccygodynia.*

This term is applied to attacks of pain in the coccygeal region, which increase on sitting, pressure, walking, and bodily exertion. It almost always occurs in women, and is attributed to a fall, injury, delivery, more rarely to a cold. In the majority of cases, the disease is probably not a neuralgia, but the result of organic changes in the coccyx. Seeligmueller produced rapid recovery in an obstinate case by the application of the faradic current. As a rule, surgical interference is necessary: subcutaneous separation of all the tendons and muscles inserted into the coccyx, or removal of the bone itself.

13. *Neuralgia of the Joints.*

1. This disease occurs generally in anæmic and hysterical women, more rarely in robust individuals or in men. Exposure, injury, acute diseases, violent emotions, diseases of the digestive tract and genital apparatus are mentioned as causes.

2. The affection is characterized by paroxysmal pain in the joints. The hip or knee joints are generally, other joints are rarely affected; sometimes the small joints, for example, of the fingers are affected. As a rule, only one joint is involved.

The pains often extend beyond the articular region proper. During the paroxysm, the integument over the joint may present redness, heat, and swelling, but these symptoms generally disappear when the pain subsides. The skin is often very sensitive to slight pressure, while vigorous pressure is often well tolerated. Muscular twitchings may occur during the attack, or the patient permanently assumes an abnormal position of the limb, which is usually kept in a position of extension (in joint inflammations the limb is usually flexed). Pressure points are found not infrequently over the joints.

If the disease lasts a long time, contracture and atrophy of the muscles may ensue.

The disease may last for years. In cases of marked hysteria the prognosis is not very good.

3. The treatment must be partly moral; we must endeavor to persuade the patient to use the limb. In addition, we may order nervines, iron, quinine, narcotics, sea-bathing, the faradic and galvanic current, etc.

b. Anæsthesia.

1. PRELIMINARY REMARKS.—Anæsthesia is the term applied to all morbid processes in the sensory nerve tracts which give rise to

diminution or complete loss of sensation. This may occur in all kinds of sensory nerves (cutaneous, muscular, visceral, etc.), but we will here discuss cutaneous anæsthesia alone.

The sensory cutaneous nerves convey two principal forms of sensations, viz.: tactile sensibility, and general sensations. Tactile sensibility includes:

a. Simple tactile sensibility, *i. e.*, the pure sensation of contact.
b. The sense of pressure.
c. The sense of location.
d. The sense of temperature.

General sensation includes the sensation of pain and electrical sensibility, also the feeling of tickling, itching, and other sensory processes which are coupled with pleasure or pain.

The methods of examination require skill and care, and the patient must possess a certain amount of intelligence and power of observation. It is best to have the patient assume a comfortable, recumbent posture during the examination, and to blindfold his eyes. He should be clearly informed as to the stages of the examination.

To determine the simple sense of contact the skin is slowly and carefully touched with the tip of the finger, or the head of a pin, and the patient directed to say whether he experiences a tactile sensation or not. The article employed should possess approximately the same temperature as the skin, since otherwise a tactile sensation may be mistaken for one of temperature.

In addition, the skin is touched with smooth, rough, and woolly substances, the character of whose surface must be described by the patient.

In examination of the sense of pressure, the limb upon which the examination is made should be placed on a firm base, since otherwise the sense of pressure would be estimated, not by the cutaneous nerves, but by the resistance overcome by the muscles. The simplest method is that of covering the integument with a disk of wood, and then placing coins upon it at regular intervals. Eulenburg devised for this purpose a baræsthesiometer, which consists of a rod movable by means of a spring, and which at the same time moves the index on a dial plate. The numbers on the dial plate indicate in grammes the amount of pressure requisite to push the index to the number in question (Fig. 28).

Fig. 28.

Baræsthesiometer.

Kaumler and Aubert found that the following were the minimum amounts of pressure which could be felt:

Forehead,			Chin,	
Temple,		0.002 grams.	Abdomen,	0.04–0.05 gm.
Dorsum of hand,			Nose,	
Forearm,			Finger Nails,	1.0 "
Fingers,		0.005–0.015 gm.		

Dohrn found that the smallest increments of weight which could be detected, if the original weight were one gm., were as follows:

Third phalanx of the fingers, 0.499 gm.	Fold of the knee	1.5 gm.
Dorsum of the foot ....0.5 "	Dorsum of the hand	1.156 "
Second phalanx of the finger..0.771 "	Forearm	1.99 "
First " " " 0.82 "	Sternum	3.0 "
Leg........ ....1.0 "	Umbilical region	3.5 "
Palm of hand.............1.018 "	Back.... ....3.8 "

Eulenburg found by means of his baræsthesiometer that the following differences in pressure could be detected:

Forehead, Lips, Dorsum of tongue, Cheeks, Temple,	$\frac{1}{40}-\frac{1}{30}$	Phalanges of fingers, Forearm, Hand, Arm,	$\frac{1}{20}-\frac{1}{10}$

In examining the sense of locality, two factors must be considered, viz., the proper localization of a stimulus, and the recognition of the tactile circle. To determine the former, an irritant is applied to some part of the skin, and the patient (with closed eyes) directed to describe the situation of the point of irritation. The measurement of the tactile circles is made by means of compasses or Sieveking's æsthesiometer. The former is an ordinary pair of compasses with blunt tips, the separation of which from one another may be read off in millimetres (vide Fig. 29). The æsthesiometer is composed of a brass rod, divided into millimetres, to one end of which is fixed a vertical arm, provided with a sharp tip, while a second one may be moved to and fro. In using the instrument (Fig. 30), both tips should be applied at the same time, and with uniform pressure. The peripheral boundaries of a tactile circle are found at those places at which the tips of the æsthesiometer are no longer felt single, but double. Their size depends upon the age of the individual, being less in childhood. Landois furnishes the following table of measurements in an adult, and a boy of twelve years.

FIG. 29.
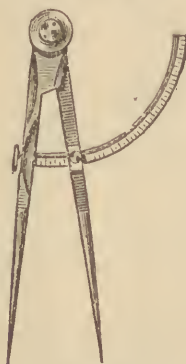
Compasses.

FIG. 30.

Sieveking's æsthesiometer.

	Adult.	Boy.		Adult.	Boy.
	Mm.	Mm.		Mm.	Mm.
Tip of the tongue..........	1.1	1.1	Skin of malar bone (posteriorly)............	22.6	15.8
Third phalanx of finger (volar).	2.3	1.7	Forehead (below)........	22.6	18.0
Lips............	4.5	3.9	Heel (behind)............	22.6	20.3
Second phalanx of finger (volar)	4.5	3.9	Occiput (below)........	27.1	22.6
Third " " " (dorsum)	6.8	4.5	Dorsum of hand.... ...31.6	31.6	22.6
Tip of nose..................	6.8	4.5	Chin (below)............	33.8	22.6
Head of metarcarpal bones (volar)...................	6.8	4.5	Vertex..................	33.8	22.6
Dorsum of tongue.... ...... } Metarcarpus of thumb...... }	9.0	6.8	Fold of knee............	36.1	31.6
Great toe (plantar)............	11.3	6.8	Sacrum ... . } Glutæi .... }	40.6	33.8
Second phalanx of finger (dorsal)	11.3	9.0	Forearm .... } Leg.................... }	40.6	36 1
Cheek	11.3	9.0	Dorsum pedis near the toes................	40.6	36.1
Lids	11.3	9.0	Sternum	45.1	33.8
Hard palate (middle)	13.5	11.3	Back of neck (high up)..54.1	54.1	36.1
Skin over malar bone (anteriorly)	15.8	11.3	Spine	54.1	..
Metastarsus of great toe (plantar)........	15.8	9.0	Middle of neck..........	67.7	..
First phalanx of finger (dorsal)	15.8	9.0	Arm .... } Thigh................ }	67.7	31.6-40.6
Head of metarcarpal bones (dorsal)....................	18.9	13.5	Middle of back........ }		
Inner surface of lips....	20.3	13.5			

In order to determine the temperature sense of the integument, Nothnagel employed round vessels of wood, with a metallic bottom, and filled with water at different temperatures. Eulenburg constructed a thermæsthesiometer. This consists of two thermometers, with large mercury bulbs, fastened to a stand upon which they can be moved to and fro. The simplest method of examination is to blow upon the skin, at the same time directing the patient to tell whether he notices the difference. Or the same part of the skin is successively touched with media at various temperatures, and the differences in sensibility noted. The ability to differentiate temperatures is most marked when the temperatures employed range about that of the body. Nothnagel furnishes the following table of the minimum of difference of temperature which can be detected in different parts of the skin:

Forearm..... .... }	0.2° C.	Palm of hand.... .... }	0.5–0.4°C.
Arm.................... ... }		Dorsum of foot .... }	
Dorsum of hand............	0.3° "	Upper part of abdomen }	
Cheek	0.4-0.2° "	(middle)............... }	0.5° "
Temple.......	0.4-0.3° "	Thigh .... }	
Chest (superiorly, exter- }		Calf...................... }	
nally).... .... }	0.5° "	Sternum .... }	0.6° "
Upper part of abdomen }		Leg (extensor side).........	0.7° "
(laterally) .... }		Back (laterally)............	0.9° "
		Back (middle).............	1.2° "

To test the sense of pain, the skin may be pricked with a pin, pinched, the hairs pulled, etc. Disturbances of the sense of touch and pain do not always go hand in hand. Retention of tactile sensation and loss of the sense of pain are known as analgesia.

Leyden and Munk also tested the electro-cutaneous sensibility. They employed a pair of copper compasses with an insulated handle, which was connected with the secondary coil of an induction apparatus, and noted the position of the cylinder when the patient experienced the first sensation, and also the first sensation of pain. Bernhardt furnishes the following table:

A. *General Sensibility of the Skin to the Electrical Current.*

Tip of tongue....	17.5 cm.	Neck at lower jaw	12.7 cm.
Palate....................	16.7 "	Arm..............	12.6 "
Tip of nose.................	15.7 "	Vertex	12.5 "
Lids.................	15.2 "	Sacrum.....................	12.35 "
Gums	15.2 "	Thigh	12.3 "
Dorsum of tongue............	15.2 "	Dorsum of first phalanx.... }	12.0 "
Vermilion of lips.............	15.1 "	Dorsum of foot........... }	
Cheeks	14.8 "	Dorsum of second phalanx...	11.75 "
White of lips................	14.5 "	Dorsum of the metacarpus.. }	11.6 "
Forehead	14.4 "	Dorsum of hand............ }	
Acromion....................	13.7 "	Leg .... }	11.5 "
Sternum............. ..... }	13.0 "	Tip of finger (volar)........ }	
Cervical spine.............. }		Tip of finger (dorsal)..........	11.3 "
Upper dorsal spine.......... }		Volar surface of metacarpus..	10.9 "
Arm........... .... }	12.8 "	Tip of toes....................	10.6 "
Buttocks .... }		Middle phalanx (volar).......	10.5 "
Middle dorsal spine......... }		Palm of hand................	10.5 "
Occiput.................... }	12.7 '	Thumb	10.5 "
Loins................ .... }		First metatarsal bone (plantar)	10.3 "

B. *Electro-Cutaneous Sensibility to Pain.*

Lids........	14.2 cm	Lower part of forehead......	12.6 cm.
Palate......................	13.9 '	Vermilion of lips........... }	12.5 "
Tip of tongue..............	14.12	Cheeks............. ....}	
Gums... ....••• }		Occiput........................	12.0 "
Tip of nose................ }	13.0	Throat below the jaw........	11.8 "
White of lips .... }		Upper dorsal spine...........	11.7 "

Middle dorsal spine...........	11.6 cm.	Dorsum of first phalanx......	9.7 cm.
Cervical spine............	11.5 "	Forearm...................	9.3 "
Sternum............... ...	11.4 "	Dorsum of metacarpus..... }	9.2 "
Acromion .... }	11.25 "	" " foot............. }	
Sacrum. .... }		" " last phalanx.	9.0 "
Loins......................	11.20 "	" " second phalanx. ..	8.7 "
Buttocks	11.1 "	Volar surface of last phalanx..	8.4 "
Dorsum of tongue...........	10.8 "	Middle of thumb.............	8.0 "
Vertex .... }		Volar surface of second	
Leg............. .... }	10.2 "	phalanx	7.9 "
Thigh..................... }		Volar surface of metacarpus..	7.6 "
Arm........	10.1 "	Palm of hand	7.5 "
Dorsum of hand	9.9 "	Tip of toes.................	6.5 "
Knee.................... ...	9.8 "	Plantar surface of metatarsus	4.0 "

The figures given in the preceding tables represent averages which may vary more or less under normal conditions. It is well to compare symmetrical parts of the body in making examinations, although it sometimes happens that both sides of the body are affected.

Total anæsthesia is paralysis or paresis of all modes of sensation; partial anæsthesia is diminution or abolition of individual modes of sensation. The various forms of sensation may be disturbed in different combinations.

Anomalous conditions of anæsthesia are sometimes observed. With regard to the sense of temperature, for example, the patients may not alone feel heat and cold less distinctly, but they may mistake heat for cold, and vice versa. Double sensations may also be felt. In tabes, for example, the prick of a pin may first produce a tactile sensation, and later a sensation of pain. Naunyn observed in tabes a double sensation of pain, the first feeble, the later one more severe, as the result of a single prick of a pin. Cases are also known in which the prick of a pin produces three or four sensations (polyæsthesia). In addition, the period between the irritation and the sensation may be unusually long (delayed sensation).

Cutaneous anæsthesia may be circumscribed or diffuse. It is sometimes confined to the distribution of one or more nerves. In spinal affections it is not infrequently distributed like paraplegia (paranæsthesia). Finally, in cerebral diseases it may be unilateral (hemianæsthesia).

II. ETIOLOGY.—The causes of anæsthesia are situated in the peripheral terminations of the sensory nerves, in those parts of the cerebral cortex in which the peripheral stimulus is converted into sensation, or in the paths of conduction between the two.

Hardly anything is known concerning diseases of the peripheral terminations of the cutaneous sensory nerves. It is true that Meissner found degenerative changes in the tactile corpuscles in cerebral diseases, but affections of the brain and cord are entirely excluded from the following considerations. Hence our chief attention will be paid to disturbances of conduction in the peripheral nerve tracts.

The sensibility of the skin depends very materially upon the normal circulation of the blood. Anæsthetic symptoms always develop in a part from which the blood has been expelled by means of an Esmarch bandage, or in which stasis has been produced by compression of a vein. This also occurs in circulatory disturbances as the result of morbid changes (arterial embolism, venous thrombosis, etc.).

Cutaneous anæsthesia is sometimes the result of thermic influences. Thus, congelation of the skin by means of ether reduces the sensibility

to such an extent that surgical operations may be performed in a painless manner. Increased heat may act in a similar manner. In some cases, probably, circulatory changes are produced chiefly or exclusively by the action of temperature; in others, perhaps, there are direct effects on the terminations of the sensory nerves.

Allied to thermal influences are the chemical noxa, which produce anæsthesia if they act upon the skin. Thus, it is well known that working in lye, strong acids, or carbolic acid rapidly produces anæsthesia.

It is also claimed that the action of the electrical current upon the skin influences the sensibility of the latter.

Toxic anæsthesia of the skin is that form which develops after the use of narcotics, when employed subcutaneously or in the form of ointment. Thus, subcutaneous injections of morphine may diminish cutaneous sensibility by direct action upon the terminations of the sensory nerves. Anæsthesia has also been observed in poisoning with lead, ergotin, or carbonic oxide, but this is perhaps the result of central causes. Anæsthesia has also been observed after infectious diseases (typhoid fever, diphtheria, syphilis, etc.).

In the majority of cases, however, morbid processes are found in the peripheral nerves (rheumatic and traumatic influences, inflammations of the nerves, compression by tumors, etc.)

III. SYMPTOMS.—Cutaneous anæsthesia is easily recognized if the methods of examination are carried out in intelligent patients. In a thorough examination, all qualities of sensation should be submitted to test. In addition, the anæsthetic district should be mapped out.

In partial anæsthesia, the symptoms may vary from time to time, inasmuch as the individual qualities of sensation may disappear or reappear at different times.

In some cases the patients call attention to the anæsthesia, because they notice diminished sensation on touching objects, or feel as if they were walking on felt or wool, etc.

If the hands are affected, the patient is apt to drop objects as soon as the eye is turned in another direction.

Some patients also complain of paræsthesia (coldness, feeling of stiffness or tension, prickling, formication).

In the simplest cases nothing is noticed beyond the objective signs of anæsthesia. In other cases, there are a number of disturbances of innervation, which depend upon an affection of other nerve tracts in addition to the sensory fibres.

For example, the most violent neuralgic pains may be felt in the affected nerve tract despite the absolute anæsthesia (anæsthesia dolorosa). Thus, a tumor which compresses a nerve may interrupt conduction from the skin to the tumor, i. e., produce anæsthesia. At the same time the tumor may irritate the central end of the nerve in such a manner as to produce in the central organ a sensation of pain which will be referred to the peripheral distribution of the compressed nerve.

Motor disturbances will be present if mixed nerves are affected, and may appear as paresis or paralysis, tonic or clonic spasms.

Reflex movements are always abolished in purely peripheral anæsthesia, simply because the stimulus is not conveyed to the cord or brain.

Vaso-motor fibres are not infrequently affected at the same time as the sensory fibres. The anæsthetic parts then present either unusual pallor, or, more frequently, unusual redness and livid discoloration of the skin; œdema and puffiness of the skin may also be noticed. This is

usually associated with changes in the cutaneous temperature (unusual heat or cold).

Anæsthesia may also be associated with trophic changes, as the result of an affection of trophic nerve fibres in the lesion. These changes include abnormal growth of hair, excessive formation and desquamation of epidermis, marked thickening and gloss of the fingers, inflammation of the nails, development of furuncles, herpes, pemphigus, extravasations, etc.

IV. Diagnosis.—As a rule, the diagnosis of cutaneous anæsthesia is not difficult. We should always endeavor to ascertain the cause and situation of the anæsthesia. An important distinction between peripheral and central anæsthesia is the absence of reflex movements in the latter. In addition, the concomitant symptoms and the distribution of the cutaneous anæsthesia must be considered in the diagnosis of the site of the lesion.

V. Prognosis.—This depends entirely on the etiology of the disease.

VI. Treatment.—This should be first directed towards the removal of the cause. In addition, we must rely on local measures, especially alcoholic and irritating inunctions, and electricity. In employing galvanism, the anode is placed on an indifferent spot, the cathode is methodically stroked to and fro over the anæsthetic region (labile application). The irritant effect is increased if the wire brush is connected with the cathode. In faradic applications, the electrodes should not be too moist, and should not be pressed too firmly upon the skin. The faradic brush may also be applied in a labile manner.

As an illustration of anæsthesia of a special nerve, we will discuss:

Anæsthesia of the Trigeminus Nerve.

I. Etiology.—The disease is not very frequent; it may affect the entire distribution or individual branches alone.

Among the causes mentioned are exposure and injury. In the former event, individual branches are alone affected. Injuries to the nerve may be of various kinds—stab and gunshot wounds, falls, blows, surgical operations, extraction of teeth, abscesses, compressing tumors, inflammations and other diseases of those bones of the skull and face through whose foramina branches of the nerve take their course; exostoses, tumors, etc., of the base of the skull, meningeal diseases, etc.

II. Symptoms.—As a rule, anæsthesia of the trigeminus is unilateral. If the entire distribution of the nerve is involved, the motor branch will also be affected, causing paralysis of the muscles of mastication on one side (vide page 13). In total trigeminal hemianæsthesia, there is loss of sensation in the face, the forehead as far as the vertex, the external, upper part of the concha, and the mucous membrane of the eye, nose, and buccal cavity. The patients suffer occasionally from paræsthesiæ or from neuralgic pains.

Circulatory and vaso-motor disturbances are not infrequent: redness and swelling of the integument of the face, changes in perspiration and the temperature of the skin.

Attention has long been attracted by certain trophic disturbances—hemorrhages and ulcerations of the buccal mucous membrane, loosening of the teeth, herpes, still more by inflammatory changes in the eye (ophthalmia neuroparalytica). In the latter cases, we find injection and

swelling of the conjunctiva, opacity and acute destruction of the cornea, followed by loss of the entire eyeball.

Meissner endeavored to show experimentally that ophthalmia neuroparalytica is due to the destruction of certain trophic fibres situated in the median part of the trunk of the trigeminus. Some authors maintained that the trophic fibres do not really belong to the trigeminus. It was shown that the ocular changes do not develop unless the nerve is injured at the Gasserian ganglion or on its peripheral aspect, *i. e.*, after sympathetic nerve fibres have joined the nerve. Numerous observers claimed, on the other hand, that the ocular affection is a traumatic keratitis, the result of the fact that, on account of the anæsthesia of the cornea, the eye is readily injured, and perhaps because, on account of interference with the movements of the lids, germs find a ready entrance into the corneal tissue.

There is sometimes anæsthesia of the entire conjunctiva and cornea, more rarely the sensibility of the cornea is retained. In the former event, touching the cornea produces neither increased secretion of tears nor winking, an important distinction from central anæsthesia. Winking will be produced if a bright light is allowed to fall upon the retina.

The application of stimuli to the nasal mucous membrane does not produce any reflex effect (sneezing); this is also true of irritation of the buccal mucous membrane. Gustatory disturbances have sometimes been observed, even though the facial nerve was intact. Hence it seems that the lingual branch of the trigeminus does not always receive all the gustatory fibres from the facial through the chorda tympani, but that the trigeminus may contain such fibres from its origin. Unilateral coating of the tongue has been described in some cases. Nothing is known concerning disturbances of salivation or audition in this disease.

If the lesion is situated at the base of the skull, the adjacent cerebral nerves, particularly the facial, acoustic, glosso-pharyngeus, and the nerves of the ocular muscles, are often paralyzed.

The duration and course of the disease depend entirely upon its causes.

III. DIAGNOSIS, PROGNOSIS AND TREATMENT.—The recognition of the disease is easy. The more the individual branches of the nerve are alone affected, the more peripherally is the lesion situated. In central anæsthesia the reflexes are always retained, if the lesion is situated on the central side of the trigeminal nuclei.

The prognosis depends on the etiology.

The treatment is the same as that of cutaneous anæsthesia in general. If the eye is affected, it must be suitably protected. Some writers claim to have obtained beneficial effects from the direct application of electricity to the eye.

C. DISEASES OF THE NERVES OF SPECIAL SENSE.

1. *Diseases of the Olfactory Nerve.*

(*Hyperosmia, Anosmia, Parosmia.*)

Very little is known concerning diseases of the olfactory nerve. Many individuals suffer from them for a long time until they are revealed by accident.

1. *Hyperosmia or olfactory hyperæsthesia* is manifested by the fact that the patients smell substances which normal individuals are unable to detect, or they are so strongly affected by certain odors that they suffer in consequence from syncope, vertigo, headache, and even general convulsions. Cases have been

known in which individuals educated the olfactory sense to such a degree that they could trace objects like dogs or birds of prey. Hyperosmia generally affects hysterical, nervous, or psychopathic individuals, and is, therefore, more often the result of central than of peripheral causes.

2. *Anosmia or olfactory anæsthesia* means the diminution or abolition of the sense of smell. In examinations those substances should be avoided which, like ammonia and acetic acid, irritate the nasal mucous membrane, and thus stimulate the trigeminus. Substances possessing a pleasant and those possessing an unpleasant odor should be employed, since the individual may be anæsthetic to one class and not to the other (partial anæsthesia). According to Althaus, irritation of the nasal mucous membrane with the constant current produces a smell like that of phosphorus (?).

Anosmic patients sometimes complain of subjective, usually unpleasant olfactory sensations; this corresponds to anæsthesia dolorosa.

Many patients also complain of gustatory disturbances. This is only true of those articles of diet whose flavor depends chiefly on the olfactory sense, for example, the bouquet of wines, the smell of cheese, meat, fruit, etc. This only occurs in bilateral anosmia.

The disease may be congenital or acquired. Cases have been reported in which the olfactory nerve was absent. Anosmia is sometimes attributed to cold, more frequently to injury. According to Ogle, a blow on the occiput may, by contrecoup, tear the fibres passing through the ethmoidal foramina from the olfactory bulbs. It is sometimes the result of nasal affections (acute and chronic coryza, polypi). Tumors, inflammations or exostoses at the base of the skull, etc., may compress the olfactory nerve and impair its function. Anosmia is sometimes associated with aphasia and right hemiplegia in disease of the left island of Reil. According to Althaus, olfactory neuritis, followed by anosmia, is a not very rare disease (?). Prevost attributes the anosmia of old age to atrophic changes in the olfactory bulbs. It has been observed occasionally after continued stimulation of the olfactory nerve, or as the result of irritating nasal injections and douches. Raynaud observed intermittent anosmia; it has also been reported after acute infectious diseases.

In certain cases the disturbance of olfaction is the result of disease of the trigeminus or facial nerve. In trigeminal anæsthesia the sensibility of the nasal mucous membrane for biting and irritating odors is impaired, and if the anæsthesia is peripheral, sneezing or other reflexes cannot be produced by tickling the mucous membrane. Olfaction proper may also be impaired by diminution of the lachrymal secretion, so that the nasal mucous membrane becomes dry and less sensitive to smells. Anosmia may also develop in facial paralysis, because paralysis of the lower lid impedes the flow of tears into the nose, and also because the paralysis of the muscles of the ala nasi impede the entrance of air into the nostril.

The prognosis is bad if the causes cannot be removed. In addition to causal treatment, the galvanic or faradic current has been applied externally to the nose or directly to the nasal mucous membrane by means of sound-shaped electrodes; strychnine has been recommended subcutaneously, internally, or locally to the nasal mucous membrane (strychnine, gr. iss.; ol. amygdal., ʒ iij.).

3. In *parosmia* there are unpleasant subjective sensations of smell, or the patient perceives unpleasant odors as pleasant, and vice versa. This is observed in hysterical and insane individuals, in epileptics as the so-called aura, sometimes in anatomical lesions of the olfactory nerves or in central diseases, if the central fibres of the olfactory nerves are affected. This group includes the predilection for certain smells which are generally regarded as unpleasant, for example, Schiller's predilection for rotten apples.

2. *Diseases of the Gustatory Nerves.*

(*Hypergeusia. Ageusia. Parageusia.*)

It is generally held at the present time that gustation upon the anterior two-thirds of the tongue is supplied by the lingual branch of the trigeminus; upon the posterior third of the tongue, soft palate, anterior palatal arch and pharynx, by the glosso-pharyngeal.

Gustatory disturbances from diseases of the glosso-pharyngeal nerve alone are unknown. We have previously shown that diseases of the facial nerve may be

associated with disturbances of taste (vide page 6). If we pass from the periphery along the course of the gustatory fibres towards the brain, the anterior two-thirds of the tongue are found to be supplied by the lingual branch of the trigeminus. The greater part of these gustatory fibres pass in the chorda tympani to the facial nerve. They again leave the facial at the geniculate ganglion, pass through the nervus petrosus superficialis major to the trigeminus, and through the spheno-palatine ganglion to the second branch of the trigeminus (vide Fig. 6). Some authors believe that the gustatory fibres in the trigeminus are derived from the glosso-pharyngeus.

The simple gustatory sensations are: sour, sweet, bitter, and salty. Sour may be tested by dilute vinegar, sweet by sugar in solution, bitter by aloes, colocynth, strychnine, salty by sodium chloride, etc. Irritating and concentrated solutions should be avoided. During the examination the patient should close his eyes, and protrude the tongue, the surface of which is touched in various places with a piece of rolled-up blotting paper, dipped in the solutions previously mentioned. A little brush or a glass rod may also be used, but the drops of fluid should not be too large.

The patient should not withdraw the tongue until he has indicated that he has tasted the substance. Before experimenting with another substance the mouth should be carefully rinsed with water. It should be remembered that bitter is perceived most distinctly by the root of the tongue, sweet by the tip, and sour by the edges.

a. In *gustatory hyperæsthesia* or *hypergeusia* the patients can taste minimum traces of a substance, or the gustatory sensations produce an unusual degree of pleasure or disgust. This is observed in hysterical and nervous individuals.

b. Gustatory anæsthesia or *ageusia* is the diminution or abolition of the sense of taste. Only a few gustatory qualities may be abolished, or in addition to ageusia, the patient suffers from perverse subjective sensations of taste. The disease may be bilateral, unilateral, or circumscribed. The conduction of the sensations is often delayed.

Ageusia is observed when the tongue is thickly coated, the buccal mucous membrane abnormally dry, and when too hot or cold substances are ingested. This is evidently the result of implication of the terminations of the lingual and glosso-pharyngeal nerves. Ageusia also occurs in diseases of the lingual nerve, and is a frequent symptom of trigeminus anæsthesia and facial paralysis. The not infrequent association of gustatory disturbances and diseases of the middle ear is effected through the chorda tympani.

Treatment consists of galvanic applications to the tongue or lingual nerve, to the petrous portion of the temporal bone, or transversely through the head.

c. Parageusia appears most frequently in hysterical and insane individuals. The gustatory sensations are generally nauseous and perverse, and may give rise to special delusions in the insane. Wernich noticed a bitter taste in the mouth after a subcutaneous injection of morphine, in feeble individuals, and in himself after prolonged fasting. Parageusia is sometimes associated with ageusia.

D. ANATOMICALLY DEMONSTRABLE DISEASES OF THE PERIPHERAL NERVES.

1. *Inflammation of the Nerves. Neuritis.*

I. ANATOMICAL CHANGES.—Inflammatory changes in the nerves may be located in the interstitial connective tissue (perineuritis) or in the nerve fibres (parenchymatous neuritis). The inflammation is sometimes confined to a circumscribed region (circumscribed neuritis) or it spreads further and further (neuritis migrans) in a centripetal or centrifugal direction. The process may spread continuously along the nerve, or it may overleap longer or shorter stretches of the nerve (neuritis disseminata).

The anatomical changes in perineuritis vary according as the disease is acute or chronic.

In acute perineuritis the nerve is unusually congested, its vessels distended, and small extravasations of blood are noticed here and there.

The surface of the nerve is less shining than usual, the inflamed part is swollen and very succulent, and the macroscopic transverse striation less distinct.

On microscopical examination the blood-vessels in the neurilemma are found unusually full, their walls are sometimes thickened and particularly shining, and the nuclei are occasionally increased in number.

Upon the outer surface of the vessels are found accumulations of round cells which are especially profuse in places. Some of these white blood-globules contain very fine granules of fat.

In some places the blood-vessels are torn, and red blood-globules are found free in the cellular tissue.

The connective-tissue parts proper of the neurilemma are swollen, and their cells increased.

If the neuritis is violent, the originally interstitial changes are complicated by parenchymatous changes, evidently as the result of the circulatory disturbances. These changes are similar to those observed in the peripheral parts of a nerve after experimental section—degeneration and disappearance of the medullary matter, followed by destruction of the axis cylinder, and marked nuclear proliferation in the sheath of Schwann. This parenchymatous degeneration is generally most marked near the perineurium.

If the acute neuritis recovers, the congestion disappears more and more, the extravasations are absorbed, the emigrated white blood-globules undergo fatty degeneration and are absorbed, and the nerve gradually assumes its normal appearance. Recovery takes place much more slowly if the interstitial process has been followed by parenchymatous changes. Very acute perineuritis may terminate in the formation of foci of pus, and these, if sufficiently large, may entirely destroy conduction in the nerve. This is most apt to occur if the perineuritis has been excited by suppuration in the vicinity of the nerve. Even gangrene has been observed under such circumstances.

Acute perineuritis not infrequently becomes chronic, or the inflammation may be chronic from the start. We then find proliferation and increased consistence of the interstitial tissue, a grayish-red color of the nerve, sometimes a grayish-black or graphite-like appearance, due to pigment left over after previous hemorrhages. The increase of connective tissue, shown by the abnormally-wide interstices, is especially distinct on transverse section of hardened nerves. The individual fibres are sometimes constricted to such an extent that they undergo atrophy, and the nerve may be converted finally into a solid band of fibrous tissue which is destitute of nerve fibres (sclerotic neuritis). Corpora amylacea are found occasionally at the site of inflammation. Chronic perineuritis may develop in a single circumscribed spot, it may spread continuously over a large tract, or it may skip over certain parts. In the latter event the inflamed portions of the nerve not infrequently form nodular or spindle-shaped prominences (neuritis nodosa). These thickenings may follow one another in the shape of a rosary. The inflamed parts of the nerve not infrequently present fibrous adhesions to surrounding parts.

Parenchymatous neuritis is rarer than perineuritis, and still rarer as an independent affection. It occurs most purely if the continuity of the nerve has been interrupted in any part of its course. The peripheral portion of the nerve then presents a series of processes which are known as degeneration, and which are partly inflammatory in their origin (neuritis degenerativa) (vide Fig. 1).

II. ETIOLOGY.—There can be no doubt that neuritis may develop as

the result of a cold, but in a much larger number of cases it is the result of injury.

Among the numerous forms of traumatism we may mention incised and gun-shot wounds, falls, blows, dislocations, fractures, compression by tumors, lifting heavy loads, etc. Neuritis sometimes develops in amputation stumps.

In some cases the inflammation extends from surrounding parts.

This is observed in pelvic abscesses, tubercular diseases of the spine, inflammation of the joints and sheaths of the tendons; after pleurisy, pneumonia, and phthisis; in progressive muscular atrophy, according to Friedreich; in cancer and sarcoma, particularly of the vertebræ. According to Leyden, many cases of reflex paralysis are the result of the extension of neuritis migrans to the spinal cord.

Neuritis sometimes follows infectious diseases (typhoid and relapsing fever, erysipelas, variola, diphtheria, syphilis, leprosy).

Toxic neuritis occurs most frequently in lead poisoning, but this form still requires careful investigation. It also develops in phosphorus poisoning.

No causes can be demonstrated in certain cases, and some authors assume a predisposition to neuritis in certain individuals.

III. PATHOLOGICAL SIGNIFICANCE OF THE DISEASE.—The experimental investigations of Tiesler, Feinberg, and Klemm have shown that neuritis may extend to and implicate the meninges and substance of the spinal cord, or that it may pass from the nerves of one side of the body to those of the other side without affecting the cord. Similar observations have been made in human beings.

It has been observed repeatedly in tetanus that neuritis disseminata, starting from a peripheral wound, has extended to the spinal cord. It is also assumed that migrating neuritis forms the connecting link in reflex epilepsy, following cicatrices in which sensory nerves are imbedded. Leyden supposes—and not without reason—that certain complexes of symptoms which have been attributed hitherto to anterior poliomyelitis are really due to extensive neuritis.

A wide field of investigation is here opened, especially since the results of experimental investigations have been disputed.

IV. SYMPTOMS.—Among the symptoms, the local phenomena play an important part from a diagnostic standpoint. They include increase in the consistence of the nerve, nodular thickenings, and pain on pressure. The pains are especially severe if the nerve is rolled to and fro between the fingers. The pain may be confined to certain parts of the nerve, or extend along its entire course. In some cases we notice a streaked or more diffuse, erysipelatous redness along the course of the nerve, with local diaphoresis and increased warmth.

The disturbances of innervation vary according as the neuritis affects a motor, sensory, or mixed nerve.

In acute neuritis of sensory nerves, tactile sensation is generally diminished in the distribution of the nerve, while pain sensation is increased (hyperalgesia). If the nerve fibres are destroyed, this is followed by loss of pain-sensation (analgesia). The patients often complain of paræsthesiæ, burning, pricking, formication, an abnormal feeling of heat or cold, etc. Spontaneous pains are almost always present. They are generally constant, but are increased on pressure, or they in-

crease spontaneously in paroxysms. Intermittent paroxysms, like those of neuralgia, are rarely observed. The pains often radiate into remote nerve tracts. Trophic changes are not infrequent: herpes zoster, pemphigus, thickening and desquamation of the epidermis, glossy fingers, changes in the nails, etc. Motor irritative symptoms (twitchings, spasms, contractures) sometimes develop as reflex symptoms.

Neuritis of motor nerves results in paresis, paralysis, tonic and clonic spasms, and contractures. These changes may undergo rapid resolution if they are the result of compression of the nerve fibres by inflammatory swelling; they are more permanent if the nerve fibres themselves have taken part in the inflammation. In the latter event, trophic changes in the muscles (diminution in the size of the individual bundles of muscular fibres, unusually distinct transverse striation, and increase of the nuclei of the sarcolemma) are apt to develop very rapidly.

At the outset, the electrical irritability is not infrequently increased, but if the inflammation is severe, the symptoms of degeneration reaction will develop (vide page 7).

Acute neuritis of mixed nerves presents a mixture of the symptoms mentioned above. Sensation is lost at an earlier period, and to a more marked degree than motion, but it returns earlier than the latter when recovery begins.

Chronic neuritis presents the same symptoms as the acute form, except that they develop more slowly and run a milder course.

The disease sometimes lasts for months and years. Recovery may be complete, or permanent disturbances may be left over.

V. DIAGNOSIS.—The diagnosis of neuritis is difficult if local changes in the nerve are absent. It may be mistaken for *a, neuralgia:* in this the pain is intermittent, and Valleix' pressure points constitute a more prominent symptom; *b, muscular rheumatism:* pain is produced by direct pressure upon the muscles; *c, embolism or thrombosis of the vessels of the limbs:* circulatory disturbances (pulselessness, œdema, cyanosis) are noticeable. *d.* The differentiation between neuritis nodosa and neuroma is not always possible, even anatomically.

VI. PROGNOSIS.—A favorable prognosis can only be given in those cases in which the disease is the result of causes which can be readily removed. But the possibility of the extension of the process to the spinal cord should always be kept in mind.

VII. TREATMENT.—Apart from causal treatment, we must rely exclusively on local measures : absolute rest, alcoholic inunctions, blue ointment, potassium iodide ointment, tincture of iodine, local warm baths, at times ice, leeches, cups, blisters, and, if the pain is very severe, subcutaneous injections of morphine. Electricity is one of the best remedies; stabile constant current, anode to the painful points, cathode indifferent or on a central painful point. We prefer feeble currents, others recommend strong currents. If the pain is very intense, good results may be obtained from the faradic brush or moxa.

In chronic neuritis we may resort to electricity, massage, nerve stretching, various baths, and cauterization.

2. *Multiple Neuritis.*
(*Progressive neuritis. Disseminated neuritis. Polyneuritis.*)

I. ETIOLOGY.—Neuritis sometimes occurs as an independent affection in many peripheral nerves, and may run an acute, subacute, or chronic course. Acute multiple neuritis may prove rapidly fatal.

The sudden onset, the rapid and febrile course of the disease raise the suspicion that we have to deal with an infectious process. Chronic forms of the disease also appear to be connected with infectious processes. For example, Scheube and Baels regard beriberi as miasmatic in its origin, and dependent on an extensive neuritis (paneuritis).

Traumatism has been mentioned as a cause of polyneuritis.

The disease has also been observed after infectious diseases, such as parotitis, acute articular rheumatism, and, according to Leyden, diphtheria, typhoid and relapsing fever, erysipelas, syphilis, and phthisis.

It generally occurs between the ages of twenty-one and thirty years, but Webber reported a case in a child of nine years.

II. SYMPTOMS.—Acute multiple neuritis is not infrequently preceded by prodromata. In one of my cases, the patient suffered from periodical chills, fever, and sweat. In other cases, there is merely a feeling of general malaise.

The manifest symptoms generally begin with pain and paræsthesiæ.

Fig. 31.
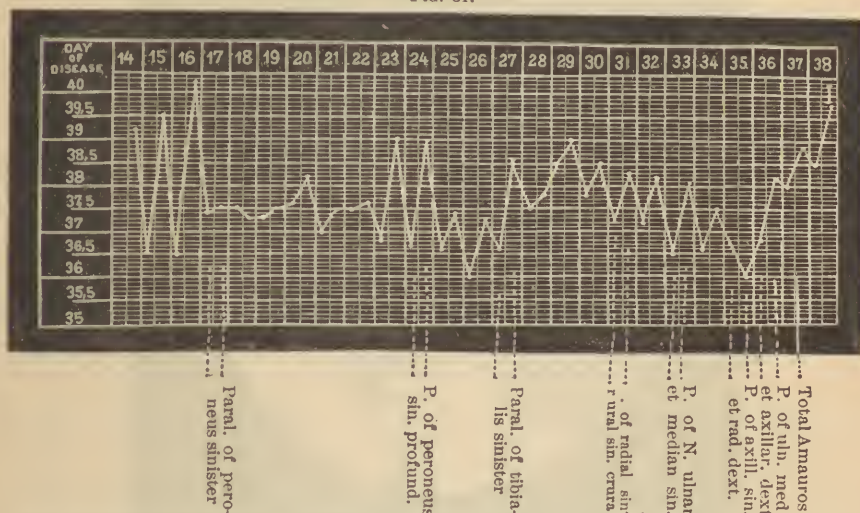
Course of the temperature and occurrence of the paralyses in acute multiple neuritis in a woman æt. 66 years.

The pain is described as burning, boring, lancinating, and may be very intense; it may be superficial or deep.

A feeling of paresis and stiffness soon becomes noticeable in the distribution of the nerve, and rapidly passes into well-marked paralysis. In my case the paralysis occurred almost as suddenly as a stroke of apoplexy.

The electrical irritability of the paralyzed muscles and nerves rapidly sinks, and degeneration reaction may be noticed within a few days.

If the paralysis does not soon recover, the muscles rapidly atrophy and later undergo contracture.

The motor nerves are chiefly, sometimes almost exclusively affected. Sensory disturbances are not infrequently present to a very slight degree. Vaso-motor changes—œdema, cyanosis and diminished temperature of the skin—are often noticed. Secretory and trophic cutaneous disturbances may also occur.

The tendon and cutaneous reflexes in the distribution of the affected nerves are lost.

The affected nerves are not infrequently tender on pressure.

The situation becomes very dangerous when the disease exhibits a tendency to spread. In a case under my observation, the symptoms began in the left peroneal nerve, then spread from one nerve to the other, from the legs to the arms, finally to the optic and pneumogastric nerves. Fig. 31 shows the temperature and date of the paralyses in this case. Implication of the vagus (shown by acceleration and irregularity of the heart's action, and by difficulty in breathing) is especially grave. Although the disease generally spreads from below upwards, this does not always hold good. It may cease at any time, and the paralysis may recover in part, while atrophy and and contracture of the muscles occurs in other parts.

The bladder and rectum are usually unaffected. Albuminuria has been observed a number of times; icterus has been described in two cases.

Fig. 32.
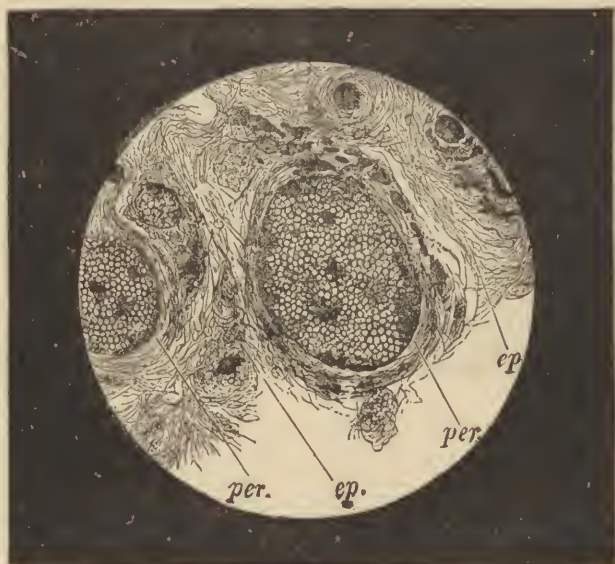
Transverse section of the left median nerve in acute multiple neuritis. Three nerve bundles are visible, with numerous extravasations of blood, partly within them, partly in the surrounding connective tissue (*per*, perineurium). Also numerous extravasations in the more remote epineurium, *ep*. Enlarged 90 times.

The symptoms of subacute and chronic multiple neuritis are similar to those of the acute form, but develop more slowly and less violently.

III. ANATOMICAL CHANGES.—We have little to add to the remarks made concerning neuritis in general.

In acute multiple neuritis, the inflammation may be chiefly interstitial or chiefly parenchymatous. In my own case, the changes were mainly interstitial. The peripheral nerves were unusually red, opaque, and succulent, and the microscope showed unusual distention of the vessels, and very numerous extravasations of blood in the insterstitial connective tissue (vide Fig. 32).

On more detailed examination, the vessels were found to be very tortuous and dilated in places, the walls were streaked, vitreous, and thick-

ened, and contained numerous endothelial nuclei; outside of the vessels were accumulations of round cells, mixed with granulo-fatty cells in addition, swelling of the connective-tissue fibrillæ and proliferation o the cellular elements of the interstitial tissue. Degenerated nerve fibre and lively proliferation of nuclei within the sheath of Schwann were found in the nerve bundles, particularly near the extravasations of blood. Mueller observed fatty changes and swelling of the paralyzed muscles.

The brain, spinal cord, and roots of the spinal nerves are intact.

IV. DIAGNOSIS.—This is not always easy. Cases which run an acute, ascending course remind us of acute ascending spinal paralysis, but the latter presents no sensory disturbances or changes in electrical irritability.

Multiple neuritis may also be mistaken for poliomyelitis; here, likewise, the presence of sensory disturbances is an important diagnostic feature.

V. PROGNOSIS.—The prognosis is always serious. In acute multiple neuritis there is danger of death when cerebral nerves, particularly the vagus, become affected. The prognosis is also serious from the fact that the paralysis often persists and cripples the patient.

VI. TREATMENT.—In the acute form salicylic acid (four to six doses of fifteen grains every quarter-hour) should be given to combat the fever and infection. Violent pains may be relieved by narcotic inunctions (for example: chloroform, ℥ iij.; liniment. volatil., ℥ iss. M. D. S. To be used t. i. d.) or subcutaneous injections of morphine. After the acute symptoms have subsided, the paralyzed muscles may be faradized and the nerves treated with the galvanic current (anode labile, cathode indifferent). If the faradic irritability of the muscles is lost, they should be treated by labile applications of the cathode of the galvanic current. Potassium iodide may be given internally. Massage and indifferent thermal waters have been employed in old cases.

PART II.

DISEASES OF THE SPINAL CORD.

A. DISEASES OF THE SUBSTANCE OF THE SPINAL CORD.

a. UNSYSTEMATIC (ATYPICAL) DISEASES OF THE CORD.

1. *Anæmia of the Spinal Cord.*

I. ETIOLOGY.—Our clinical knowledge of this disease is very scanty. Its causes may be local or general. The former include: compression, embolism, or thrombosis of the abdominal aorta, embolism or thrombosis of fine vessels in the spinal cord, and compression of the cord. The general causes include: profuse losses of blood, chlorosis, protracted suppuration, etc.

Brown-Séquard attributes reflex paralysis to spasm of the arteries in the spinal cord.

In 1667, Stenson produced paralysis of the muscles, sphincters, and sensation by compression of the abdominal aorta in animals (anæmia of the spinal cord). Panum produced embolism of spinal vessels by injecting substances into the blood.

II. ANATOMICAL CHANGES.—The substance of the spinal cord is pale, and the gray matter is depressed below the white matter. The latter is softened, more

rarely its consistence is increased. Red and yellow softening is sometimes observed as an expression of anæmic necrosis of the cord. If the anæmia is the result of compression or embolism of small vessels in the cord, the periphery of the anæmic focus will be marked by unusual congestion of the vessels and extravasations of blood. Leyden's observations indicate that microscopical examination is necessary to detect fine emboli in the cord.

The meninges are also anæmic, but the venous plexuses in the peridural cellular tissue are occasionally distended with blood.

We must be on our guard against mistaking these appearances for cadaveric changes in the cord, particularly if no causes of anæmia are demonstrable.

III. SYMPTOMS.—If anæmia of a large part of the lumbar cord suddenly occur, for example, as the result of embolism of the abdominal aorta, it will be followed by rapid paralysis of the lower limbs, loss of sensibility and reflex irritability, and paralysis of the bladder and rectum. At the same time the pulse in the crural arteries disappears or becomes very small, and the limbs undergo gangrene unless the collateral circulation is quickly re-established.

Embolism of smaller vessels of the cord may not give rise to symptoms.

To spinal anæmia as the result of general anæmia have been attributed conditions of paresis, paralysis, tremor, hyperæsthesia, anæsthesia, and increased reflex excitability. Such conditions may disappear as soon as the general anæmia is relieved.

IV. DIAGNOSIS.—The diagnosis is not always easy; it depends particularly upon the demonstration of anæmia as a cause of the condition.

V. PROGNOSIS.—The prognosis is unfavorable if nutritive disturbances occur in the cord and are followed by symptoms of organic lesions.

VI. TREATMENT.—This is chiefly causal and symptomatic. Brown-Séquard recommends dorsal decubitus with elevated head and limbs. Bags of hot sand or hot water may be applied to the back. Strychnine, opium. and amyl nitrite have been recommended. In other respects, purely symptomatic treatment.

2. *Hyperæmia of the Spinal Cord.*

I. ANATOMICAL CHANGES.—The term congestion of the cord is often employed by physicians who are anxious to make fine diagnoses, but our knowledge of this condition is almost nil.

On transverse section of the congested cord, the white substance has a rosy red appearance, presents numerous dots of blood, unusually large and sinuous vessels, in places true extravasations. The gray matter has a red, sometimes almost brownish-red color. The consistence of the cord may be increased or diminished. The meninges also appear congested, and the veins of the peridural tissue are sometimes sinuous and dilated. The spinal fluid is not infrequently increased. sometimes cloudy, occasionally tinged with blood.

Congestion and even hemorrhages of the nerve roots have been noticed in some cases.

Hyperæmia of the cord may have been present during life, although the appearances have disappeared in the dead-house. The reverse also holds good, *i. e.*, congestion of the cord may appear to be present after death, either as the result of prolonged position on the back, or cadaveric decomposition, etc.

II. ETIOLOGY.—Active spinal congestion may be the result of cold, shock, inflammation, infectious diseases, bodily exertion, onanism, excessive coitus, poisoning with carbonic oxide, strychnia, alcohol, prussic acid. It may also develop in congestive conditions of certain abdominal organs, for example, in menstrual and hemorrhoidal hemorrhages, or in the absence of customary hemorrhages of this character.

Passive spinal congestion may be the result of general disturbances in cardiac and pulmonary diseases. Or the causes of stasis may be local stasis of the portal vein, abdominal tumors, pregnant uterus. This also includes spasmodic conditions (epilepsy, tetanus, uræmia, agonal spasms, etc.) in which congestion and hemorrhages, particularly into the peridural tissue, often occur, and were often falsely regarded as the causes of these diseases.

III. SYMPTOMS.—The symptoms of active and passive spinal congestion are alike; the lower limbs are generally affected. Among the symptoms mentioned are: a feeling of pressure, tension, and pain in the spine on external pressure, a cincture feeling around the belly, pains in the limbs, hyperæsthesia, paræsthesia, rarely anæsthesia, twitchings in some of the muscles, rarely complete paralysis, increased reflex excitability, and galvanic irritability. The bladder and rectum

are rarely paralyzed. If the upper cervical cord is affected, there may be disturbances of respiration and cardiac action. Cerebral congestion may also be present, and its symptoms may occupy the foreground. According to Brown-Séquard, the symptoms increase in dorsal decubitus, decrease in abdominal decubitus or in the standing position. Others maintain that the symptoms increase in the erect posture. They often vary with remarkable rapidity.

IV. TREATMENT.—We may apply cups or leeches (ten to twenty) to the spine, or apply leeches to the anus or cervix uteri, ice bags to the spine, foot or hand mustard baths; internally ergotin or belladonna. The patient should assume abdominal or lateral decubitus, and the bowels should be thoroughly evacuated daily.

3. *Hemorrhage of the Spinal Cord.*

(*Spinal Apoplexy. Hæmatomyelia.*)

I. ETIOLOGY.—Primary hemorrhages into the cord are rare, and Charcot and Hayem deny their occurrence. At all events secondary hemorrhages are much more frequent, especially in connection with previous inflammation of the spinal cord (hæmatomyelitis).

Among the causes of primary spinal hemorrhage are injuries, cold, bodily strain, coitus, sexual excesses in general, stasis following cardiac, pulmonary, and hepatic diseases. Previous disease of the spinal cord or the immediate vicinity also favors the development of spinal hemorrhage.

Males are affected more frequently than females.

II. ANATOMICAL CHANGES.—The hemorrhagic focus in the cord cannot infrequently be recognized beneath the pia mater, as soon as the dura mater has been laid open. It often shows through the pia mater as a bluish-black mass, and sometimes causes a prominence of the meninges. The pia mater may even be ruptured, and the blood escape into the subarachnoid cavity.

The upper cervical or the dorsal portions of the cord are most frequently affected. There is usually only one extravasation. It almost always begins in the gray matter, particularly the anterior horns, and not infrequently occupies the entire section of the cord. In rare cases the blood extends along the length of the cord, following the longitudinal fibres in the white matter, and attains a length of more than ten cm.

Recent hemorrhages have a blackish-red, older ones a brownish-red, rusty brown, even ochre-yellow color. In recent foci the blood is partly coagulated, and the adjacent nerve tissue is generally in a condition of red or yellow inflammatory softening. After a while a sort of fibrous capsule may form around the hemorrhage, and the blood is replaced by serous fluid (so-called apoplectic cysts). Smaller extravasations generally terminate in a sort of connective tissue, pigmented cicatrix. Spinal hemorrhages may not alone be followed by meningeal hemorrhages, but occasionally by fibrous and even purulent inflammations.

Among the complications and sequelæ are inflammatory changes and secondary degeneration of the cord (vide the section on secondary spinal degenerations).

III. SYMPTOMS.—The disease is characterized by the sudden (apoplectiform) occurrence of paralytic symptoms, which vary according to the situation and extent of the hemorrhage. The symptoms generally correspond to those of complete section of the cord, sometimes to those of a unilateral lesion. In other cases they indicate exclusive or chief implication of the anterior, more rarely the posterior horns.

As a rule, the symptoms occur suddenly without prodromata, more

rarely they are preceded for a few hours or days by morbid phenomena, which indicate congestion of the cord and meninges (pain in the back, stiffness in the spine, paræsthesiæ in the limbs, etc.).

Consciousness is not impaired at the onset, unless the hemorrhage is situated high up in the cervical enlargement of the cord. In some cases the symptoms of shock are manifested.

In the beginning, the bodily temperature is unchanged; in the next few days there is sometimes a slight rise of temperature. High fever develops in the later course of the disease if it becomes complicated with cystitis, ammoniacal decomposition of the urine, and bed-sores.

The majority of patients complain at first of more or less violent pain in the spine, corresponding to the site of hemorrhage, but the pain generally subsides in a few hours.

The pain is usually associated with paralysis, as a rule paraplegia, more rarely spinal hemiplegia or monoplegia. The paralysis is often complete from the beginning, in other cases it is incomplete at first, but increases in severity during the next few hours. It affects motion and sensation, the bladder and rectum. The reflexes are often abolished or enfeebled, but, if the cord has been completely cut across, the reflex irritability below the site of lesion will be increased at the end of a few days. The reflexes will remain absent permanently if an extensive hemorrhage has taken place into the lumbar part of the cord, or if the anterior or posterior nerve roots are affected (in the latter event the reflexes will be abolished merely in the distribution of the affected nerves). Destruction of the anterior horns of gray matter or the anterior roots is shown by rapid atrophy of the paralyzed muscles, and the development of the degeneration reaction. Spontaneous twitchings are sometimes observed in the paralyzed muscles, probably as the result of irritative conditions below the site of hemorrhage. Vaso-motor and trophic disturbances in the paralyzed limbs have also been described. Hæmaturia and albuminuria often develop in a short time.

Death may follow almost instantaneously if the hemorrhage is situated so high as to involve the medulla oblongata and its respiratory centre. In hemorrhages near the cervical enlargement, the symptoms of paralysis of the diaphragm may occur (affection of the phrenic nerve) and produce fatal suffocation. The disease may also be protracted for weeks, months, and even a couple of years. In certain cases, acute decubitus or cystitis and ammoniacal decomposition of the urine may give rise to septicæmic symptoms, increasing exhaustion, and death.

IV. DIAGNOSIS.—The chief interest attaches to two factors in diagnosis—to the sudden occurrence of the symptoms, and to the predominance of paralytic phenomena. Although meningeal hemorrhage may occur with equal suddenness, irritative symptoms, viz., pain, muscular twitchings, etc., predominate in this disease. In hemorrhagic central myelitis, extensive paralysis may likewise develop in a very short time, but the spread of the process can usually be made out with distinctness. Acute anterior poliomyelitis begins with fever, the paralytic symptoms do not develop so suddenly, and sensory disturbances, paralysis of the bladder and rectum, and decubitus are absent. Cerebral hemorrhage is readily excluded by the absence of paralysis of cerebral nerves, particularly of the facial nerve; in addition, the paralysis is generally hemiplegic.

The recognition of the site of hemorrhage is usually easy. It may often be suspected from the location of the pains or cincture feeling. If

the lumbar cord is involved, we generally find paraplegia of the lower limbs, paralysis of the bladder and rectum, and loss of reflex irritability; priapism is sometimes noticed. In dorsal hemorrhage, the symptoms remain the same, except that reflex movements reappear or are increased, and that the abdominal muscles are paralyzed. Cervical hemorrhage causes paraplegia of all the limbs, and sometimes paralysis of the phrenic nerve. Bulbar symptoms (pupillary changes, slowness or acceleration of the pulse, difficulty in deglutition, etc.) supervene if the hemorrhage is situated still higher.

V. Prognosis.—The prognosis is unfavorable, since permanent paralyses are left over, even if the clot is absorbed. Death may occur under different circumstances with varying rapidity.

VI. Treatment.—If there are symptoms of spinal congestion, ten to twenty leeches or cups should be applied to the spine, or five to ten leeches to the anus or cervix uteri. Daily evacuations from the bowels should be secured. The patients should be kept as quiet as possible, upon a Chapman bag filled with ice or a water-bag. Potassium iodide (℥ iij. : ℥ iij., one tablespoonful t. i. d.) should be given internally, to facilitate absorption of the extravasation. After the cessation of the acute symptoms (in six to ten weeks), the galvanic current may be applied (at first the anode, then the cathode) stabile to the site of the disease, for five minutes three times a week, the indifferent electrode on the sternum.

The Chapman rubber-bag is extremely important in the treatment of diseases of the cord. It consists of three long rubber-bags, each of which is larger than the one above it (vide Fig. 33). The bags are filled with ice until the latter reaches to the lower end of the adjacent bag.

In order to prevent inflammation of the skin, a piece of linen should always be interposed between the rubber-bag and the integument. The bag should be refilled as soon as the ice has melted.

Fig. 33.

Chapman's Rubber Bag.
¼ natural size.

Appendix.

Capillary (punctate) hemorrhages of the spinal cord usually possess no clinical significance. Paralytic symptoms can only be expected when they are very numerous and closely aggregated. The hemorrhages are found not infrequently on a transverse section of the cord, either exclusively or chiefly in the gray matter. They are rarely larger than a fine dot. Unlike bleeding vessels, they cannot be removed by a stream of water or by the fingers. They are situated in the lymphatic sheaths of the vessels, which are sometimes ruptured, so that the blood is free in the tissues.

The hemorrhages often constitute an agonal symptom, and are also found in spasmodic diseases, in which they may be associated with meningeal hemorrhages. They are often found in infectious and severe constitutional diseases.

These hemorrhages have also been produced experimentally in animals which were placed in rarefied air; they were so numerous as to give rise to paralytic symptoms. Similar conditions have also been observed in men who worked for

6
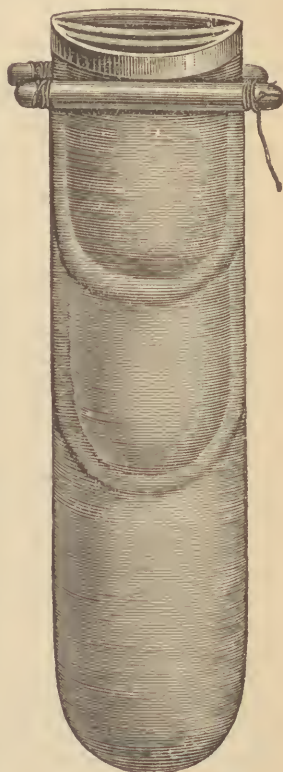
a long time in compressed air and then passed into the general atmosphere. In these cases, however, hemorrhages were not found in the spinal cord. In a case of this kind, Leyden found numerous cavities in the cord surrounded by large cells, which he regards as altered white blood-globules. He explains the appearances by Hoppe's experiments, according to which, in animals placed in rarefied air, the gases partly escape from the blood and produce further serious changes.

4. *Acute Inflammation of the Spinal Cord.*

Acute Myelitis.

I. ETIOLOGY.—Acute myelitis is one of the rarer diseases. It is relatively frequent in middle life (fifteen to thirty years), and more frequent in men than in women.

A predisposition to the disease is engendered by alcoholism, dissipation, overwork, living in damp rooms, etc. Under such circumstances myelitis may be produced by very slight causes.

Myelitis may be primary or secondary. The former includes rheumatic, traumatic, and toxic forms; it may also be the result of violent mental excitement. Some claim that the disease may follow excesses in venery, onanism, cessation of menstrual and hemorrhoidal hemorrhages, but this is not beyond dispute.

There can be no doubt that a cold is a not infrequent cause of myelitis; it occurs occasionally after a wetting, sleeping on the damp ground, etc.

Traumatic myelitis is still more frequent than the rheumatic form. It is observed after wounds of the cord, fracture and dislocation of the vertebræ, even after violent concussion of the spine or of the entire body.

Toxic myelitis is better known experimentally than clinically. It has been produced in animals by poisoning with phosphorus, arsenic, mercury, lead, carbonic oxide, and alcohol.

Secondary acute myelitis often follows diseases of the vertebræ or meninges (tuberculosis or cancer of the vertebræ, inflammations, hemorrhages, and tumors of the meninges). It is sometimes a sequel of neuritis migrans, occasionally of previous diseases of the spinal cord itself.

It may also develop during or after certain acute and chronic infectious diseases, viz , diphtheria, pneumonia, typhoid fever, articular rheumatism, pyæmia, puerperal fever, malaria, and especially syphilis. Perhaps certain cases of myelitis are associated with pulmonary phthisis.

II. ANATOMICAL CHANGES.—So far as is known, the process begins with changes in the interstitial tissue (vessels and neuroglia), and these are immediately followed by an affection of the nervous tissue proper. The dorsal cord is affected most frequently, next the lumbar and cervical portions.

The first changes almost always occur in the gray substance of the cord (central myelitis), to which it may be confined; in other cases it extends to the white matter. Sometimes the entire transverse section of the cord is inflamed (transverse myelitis), sometimes there is only a small spot of inflammation (circumscribed myelitis), which is perhaps confined to one-half of the cord. In certain cases there are numerous foci of inflammation which may be so small as to be recognizable only under the microscope. In rare cases the inflammatory changes are confined to the periphery, and are associated with meningeal changes (perimyelitis or myelo-meningitis).

When acute myelitis attains certain dimensions, the color and con-

sistence of the parts are changed. Their consistence is diminished, though softening of the cord may also be non-inflammatory in character. Thus we sometimes find post-mortem softening of the cord, particularly in the dorsal portion, in bodies which have lain on the back for a long time during the summer months. Hence other changes must also be present in order to prove the inflammatory character of the softening. Upon section, the softened parts project above the cut surface and form a more or less soft pulp; in some cases they are entirely fluid. The normal markings are indistinct or entirely obliterated. According to the duration of the inflammation, the softened parts present a different color (red, yellow, gray softening).

In red inflammatory softening of the cord, the parts have a bright-red, brownish-red, or chocolate color. Numerous extravasations are often found in the surrounding parts, sometimes in the inflamed parts themselves (hemorrhagic or apoplectiform myelitis).

Gradual absorption and transformation of the blood pigment and increasing fatty degeneration of the constituents of the cord give rise to yellow softening. Finally the pigment disappears in great part, the fatty elements are absorbed, resulting in gray softening.

Microscopical examination should be made, partly on the fresh, partly on the hardened cord. In the former method, scrapings from the knife-blade may be examined in a one-half-per-cent solution of sodium chloride, or in diluted glycerin. The cord may be hardened in potassium bichromate or ammonium chromate, the dura mater having first been opened and the cord cut into sections three to five cm. in length. The cord is placed in a one-per-cent solution of the hardening fluid for one week, and the strength of the fluid increased one per cent weekly for a month. It becomes fit for making sections after it has been eight to twelve weeks in the five-per-cent fluid.

The first microscopical changes in acute myelitis appear in the blood-vessels and neuroglia. The vessels, especially the small veins, are unusually dilated, sometimes very sinuous. Their walls are peculiarly thickened and shining, occasionally fibrillated, and the nuclei increased in number. The lymph sheaths are unusually wide, and contain red or white blood-globules. Extravasations may also occur through these sheaths themselves, and appear upon their outer surface.

At the same time, changes develop in the neuroglia; its cellular constituents increase in size and number, and their nuclei also proliferate. Some of these cells are perhaps emigrated leucocytes. The basement substance of the neuroglia is swollen.

These lesions are soon followed by changes in the nerve fibres and ganglion cells. The medullary substance of the fibres coagulates, becomes finely granular, takes carmine staining, and finally disappears entirely. At the same time the axis cylinders undergo striking changes in shape. In places they are enormously distended into a spindle-shape; several of these enlargements sometimes follow one another, like a rosary. At the same time, the axis cylinder becomes homogeneous, vacuolæ form in it, and nuclear proliferation has also been described. Next it becomes granular, and later is absorbed.

The ganglion cells lose their granular and fibrillated structure, and assume a swollen, homogeneous, and vitreous appearance. They often contain vacuoles. The prolongations are thickened and often separated from the cells. The nucleoli become indistinct, but sometimes proliferate; the nucleus finally becomes more and more indistinct. The cell substance becomes more granular and fatty, and is absorbed; the affected cells often contain an abnormal amount of pigment. Some of the ganglion cells do not disappear, but are converted into small round bodies without prolongations which may remain for a long time in that condition. Foerster observed calcification of the ganglion cells.

An important phenomenon is the abundant development of granulo-fatty cells, which are found not alone in inflammation, but also in necrotic processes. These cells are round, composed of fine granules of fat, sometimes contain one or more drops of myelin, and generally disclose a nucleus after staining with carmine.

In hardened preparations they are detected most readily after treatment with potash.

These granulo-fatty cells are present in remarkable numbers. They are partly free in the interstices, partly in the lymphatic sheaths of the vessels. In our opinion, they play an important part in the absorption of the fatty masses which result from degeneration of the nerve fibres and ganglion cells. To render the absorption of the fat possible, wandering cells are requisite, which will become stuffed full with fat, and then make their way into the lymphatic sheaths of the vessels.

In addition to the granulo-fatty cells, we also find laminated amyloid bodies, whose origin and significance are unknown.

In some cases a sort of coagulated exudation is noticeable. It forms vitreous, colloid clumps which are situated partly in the lymphatic sheaths, but in great part upon the external wall of the blood-vessels, sometimes free in the interstices.

The extravasated red blood-globules are gradually destroyed, their pigment is deposited in granules or crystalline form, or it is imbibed by the granulo-fatty cells or by larger drops of fat.

If the inflammation is very intense, it may terminate in the formation of a circumscribed deposit of pus in the cord (abscess of the spinal cord). This rare occurrence is observed most frequently when the myelitis is traumatic in origin, and foreign bodies have remained in the cord.

If the inflammatory process is protracted, it may terminate in the formation of cysts. The softened parts of the spinal cord become more and more fluid, forming at first an emulsion-like, then a milky, finally a serous fluid. At the same time the surrounding neuroglia proliferates so that a fibrous capsule forms around the fluid. The cyst is often multilocular.

In some cases the originally softened spot in the cord undergoes sclerosis, and the disease then usually becomes chronic. The indurated tissue is composed chiefly of new-formed neuroglia which is rich in cells, contains few nervous elements, granulo-fatty cells, and corpora amylacea.

Dujardin-Beaumetz has applied the term hyperplastic myelitis to a certain anatomical form in which the interstitial inflammatory changes are especially marked, and the inflamed parts are firm to the feel.

The ascending and descending secondary degenerations will be discussed in a subsequent chapter.

As a rule, the meninges, particularly the pia mater, also take part in the inflammation. The pia mater is reddened, succulent, occasionally infiltrated with pus. According to Schultze, acute myelitis following syphilis is characterized by marked affection of the meninges and vascular apparatus.

The nerve-roots have been found reddened and swollen, and sometimes present spindle-shaped enlargements; nuclear proliferation and degeneration of the nerve-fibres may be seen under the microscope.

Degenerative changes have also been observed in the peripheral nerves; increase of the nuclei, unusually distinct transverse striation, more rarely fatty degeneration in the muscles, have been described.

Among other organic changes in acute myelitis may be mentioned : bed-sores; inflammation, hemorrhage, ulceration and partial desquamation of the mucous membrane of the urinary passages, particularly the bladder and pelvis of the kidney; decomposed urine in the bladder; not infrequently multiple abscesses in the kidneys; hemorrhages into the suprarenal capsules.

III. SYMPTOMS.—The symptoms of acute myelitis may begin suddenly or may be preceded by prodromata for a few hours or days. The prodromata are often of a general, indefinite character: chilly sensa-

tions, elevation of temperature, anorexia, increased thirst, general malaise, etc. The symptomatology is sometimes like that of an acute infectious disease; it begins with a chill, followed by high temperature, and then the other manifest symptoms of myelitis develop rapidly and unmistakably. In children general convulsions may occur.

The suspicion of an acute spinal affection must be aroused if the symptoms described are followed by disturbances of innervation, such as formication in the limbs, a feeling of coldness, shooting pains, etc. Retention of urine, more rarely incontinence of the bladder, is occasionally one of the first symptoms.

Local changes soon become noticeable. The patients complain of pain in a circumscribed part of the spine, corresponding to the site of disease. The pains are sometimes spontaneous, or they are provoked by movement of the spine, pressure, etc. Many patients complain of a constricting, band-like feeling which extends from the painful spot around the abdomen or thorax. Cardialgic symptoms sometimes appear at an early period.

In the mean time, the disturbances of innervation have become more distinct. Irritative symptoms (pain, hyperæsthesia, twitchings, and contractures of the muscles) are temporary in character, and generally present only at the beginning of the disease, while, as a rule, paralyses constitute the chief feature of the symptomatology. The pains are usually neuralgiform in character; they are sometimes localized in certain parts, for example, the joints. The pains are sometimes present after the sensory and motor nerves are completely paralyzed (anæsthesia dolorosa).

Paralysis of the muscles is one of the chief symptoms of acute myelitis. It may be complete or incomplete, but the paralyzed parts are always flaccid. Transverse myelitis of the lumbar cord gives rise to paraplegia of the lower limbs, in addition to paralysis of the bladder and rectum. In a similar affection of the dorsal cord, the abdominal muscles are also paralyzed, so that the patients cannot cough and strain. Under such circumstances, even a mild bronchitis may be a source of danger. In myelitis of the cervical enlargement, the arms and chest muscles are also paralyzed. Hence inspiration is interfered with to such an extent that diaphragmatic breathing becomes unusually vigorous. In cervical myelitis, the paralysis is sometimes confined to the arms, while the legs escape. Inflammation of the upper part of the cord is shown by implication of bulbar nerves: unilateral or bilateral myosis, slowness of the pulse (twenty-eight beats in a minute in one case), disturbances of deglutition, difficulty in articulation, etc. Redness and increased heat of the face and neck indicate paralysis of the sympathetic. Descending optic neuritis, with subsequent atrophy, has also been described. Painful attacks of palpitation of the heart are sometimes observed. The chief danger in cervical myelitis arises from paralysis of the phrenic nerve; respiration is then impeded, and death takes place from asphyxia.

The nutrition of the paralyzed muscles may be unimpaired for a long time, until atrophy from disuse gradually develops. But in some cases there is rapid emaciation, as the result of destruction of the large (trophic) cells in the anterior horns of the gray matter, or of implication of the nerve-roots in the morbid process.

In the latter event, the degeneration reaction is produced in the paralyzed muscles. In atrophy from disease, the electrical irritability

gradually diminishes to a slight extent, and to an equal degree to both currents.

At first the cutaneous sensibility is occasionally diminished or abolished only for certain modes of sensation; later, complete anæsthesia generally develops. In the former event, delayed conduction is sometimes noticeable. Certain patients experience a sensation of vibration of the entire limb after the application of circumscribed stimuli, for example, the prick of a pin (dysæsthesia).

Vaso-motor disturbances are almost always noticeable. The paralyzed limbs are reddened or livid, warmer than normal, and do not sweat. Brieger found that the paralyzed parts remained dry even after diaphoresis was produced by injections of pilocarpine. After the disease

Fig. 34.
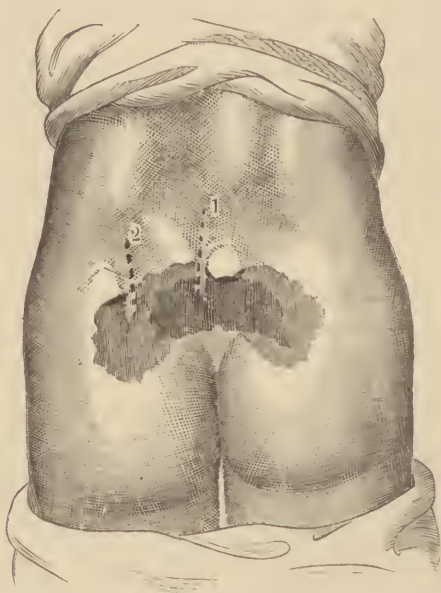
Acute decubitus in dorsal myelitis. 1, gangrenous; 2, erythematous portion. After Charcot.

has lasted for some time, the paralyzed parts become pale and cold. Œdema of the skin and swelling of the joints are not infrequent.

Nor are trophic changes rare. They consist of active desquamation of the epidermis, erythema, vesicular eruptions, and especially acute decubitus. This develops most frequently over the sacrum, next over the trochanters, malleoli, and heels. The skin reddens, then vesicular elevations form, and finally the tissues undergo rapid gangrenous destruction. This often spreads with terrible rapidity, extends through the spinal column, and invades the meninges and spinal cord. The development of decubitus is favored by pressure on the parts, but it may also develop independently, as the sole result of trophic disturbances. It has been attributed to disease of the posterior horns and the adjacent parts of the posterior columns of the cord.

Reflex irritability is affected in various ways. If the inflammation extends through the lumbar cord, reflex action in the lower limbs is necessarily abolished. If the lesion is situated higher, the reflex irritability of the lower limbs may be diminished or abolished at first as the result of the severe general disturbance of the cord, but later the reflex irritability is increased and does not disappear again until the inflammation has assumed larger proportions. The cutaneous and tendon reflexes are affected in the same way.

Important changes in the urine very often develop at an early period, and it has been assumed by some that these are the result of trophic changes, not of simple stagnation of urine. The urine becomes bloody, albuminous, cloudy, and ammoniacal, and the retention of urine may give rise to septicæmic and pyæmic conditions. Engelken observed sugar in the urine in dorsal myelitis.

The bladder and rectum are paralyzed from the beginning (in lumbar myelitis), or not until a later period of the disease. As a rule, there is at first retention of urine (paralysis of the detrusor vesicæ), and incontinence occurs at a later period (paralysis of the sphincter vesicæ). Or paralysis of both muscles occurs simultaneously, i. e., there is a constant flow of urine, although the bladder is not thoroughly emptied. A similar condition obtains with regard to the rectum—at first obstinate constipation, later involuntary evacuations. In cervical and dorsal myelitis, priapism is not infrequent; the erection is generally flaccid, but painful. Involuntary discharge of semen has also been described.

The paralysis sometimes begins suddenly at night, and within a few hours or days spreads upwards or downwards, or in both directions, disturbances of innervation on the part of the medulla set in, the temperature of the body often becomes hyperpyrexial, and finally death occurs from the excessive rise of temperature or from suffocation. This is especially apt to occur in hemorrhagic myelitis.

In other cases, the disease lasts one to three weeks; exhaustion, decubitus, ammoniæmia, septicæmic and pyæmic conditions, or a complicating pneumonia put an end to life.

In still other cases, the disease runs a subacute course, and lasts many weeks.

French writers apply the term recurrent myelitis to a form which is characterized by relapses, the intervals sometimes lasting several months.

If the disease becomes chronic, certain paralyses disappear, others remain permanent. Recovery of the paralysis is possible in those cases in which certain parts of the cord are compressed by inflammatory swelling or œdema, and this pressure is relieved. Muscular twitchings and contractures, and exaggeration of the tendon reflexes indicate secondary degeneration in the cord.

Complete recovery can hardly be looked for.

IV. Diagnosis.—The recognition of acute myelitis is usually easy. It is distinguished from acute spinal meningitis by the fact that in the latter the irritative symptoms are more marked, the paralysis less marked. In spinal and meningeal hemorrhage, the symptoms begin more suddenly, and in the latter affection there are marked symptoms of irritation; in addition, a greater degree of recovery is possible. Ascending myelitis may be mistaken for Landry's ascending paralysis, but in the latter, the electrical excitability is unchanged, and the course of the

disease is usually more. rapid. In multiple neuritis, the electrical irritability of the paralyzed nerves and muscles rapidly diminishes. The differentiation from hysterical paralysis is sometimes difficult, but this disease generally affects women, and other symptoms of hysteria are usually present.

We should also endeavor to ascertain the height of the morbid process. We must therefore take into consideration the painful points on the spine, the distribution of the motor paralysis, and particularly the extent of the cutaneous anæsthesia. If the latter is tested from below upwards, the boundary of the anæsthetic region will be found a little below the site of inflammation in the cord. In lumbar myelitis, the anæsthesia usually extends about to the level of the umbilicus. In myelitis of the lower dorsal cord, the skin is anæsthetic as far as the lower end of the sternum. In myelitis of the upper part of the dorsal cord, the anæsthetic zone extends to the axilla; in cervical myelitis it involves the upper limbs.

The following statements may be made concerning the extension of the inflammation over the transverse section of the cord. Motor paralysis indicates an affection of the lateral columns, particularly the innermost parts and adjacent gray substance (pyramid tracts). If the motor paralysis is followed by rapid atrophy and degeneration reaction in the paralyzed muscles and nerves, we may assume destruction of the anterior horns (poliomyelitis) or interruption to conduction in the anterior roots. The latter is more probable if marked cincture feeling and pains are noticed. Sensory disturbances indicate implication of the posterior columns, posterior horns, or posterior roots.

V. Prognosis.—The prognosis is almost always unfavorable as regards recovery. The greater the tendency to spread transversely or along the cord, the more unfavorable is the prognosis. Decubitus, alkaline decomposition of the urine, paralysis of the phrenic, and bulbar symptoms are especially grave symptoms.

VI. Treatment.—Causal treatment should be adopted if the acute myelitis is the result of syphilis. We may then order inunctions of mercurial ointment (gr. lxxv. daily) and potassium iodide internally (gr. xij. t. i. d.). Some authors recommend the internal administration of calomel. Mercury has also been recommended as an active antiphlogistic even in cases of non-syphilitic origin.

In the majority of cases, the treatment must be purely symptomatic. The patient's bed should be perfectly smooth, in order to avoid pressure and thus prevent bed-sores. He should lie upon the side or abdomen as much as possible, and frequently change the position of the body. In addition, the skin should be rubbed daily with alcohol in order to harden it. If the skin grows red or decubitus begins, the parts should be covered with adhesive plaster which is renewed daily. In addition, an air bag should be used. But if the bed-sore has already spread widely, the best services are rendered by the permanent water bath (28° to 30° R.).

In retention of urine, the bladder should be catheterized three times a day, care being taken to keep the catheter scrupulously clean by keeping it constantly in a five-per-cent solution of carbolic acid. If the urine is constantly dribbling away, a rubber urinal may be given to male patients, while females may use a bed-pan encased in rubber. In our opinion, the permanent introduction of a catheter into the bladder is

less advisable. We should always endeavor to avoid soiling the skin with urine, since this is apt to result in decubitus.

If the urine has undergone decomposition, it may be caught in vessels in which fifteen to seventy-five grains of naphthalin have been placed to correct the urinous odor. We have also seen good results from the internal administration of naphthalin (gr. vij. every two hours), but the remedy must be discontinued in some patients because it produces intolerable burning in the urethra, and even albuminuria. We should then order potassium chlorate, salicylic acid, etc., and wash the bladder at regular intervals (vide Vol. II., page 122).

If the patients suffer from constipation, we may employ the remedies recommended in Vol. II., page 334; if incontinence of fæces occurs, a bed-pan should be used at regular intervals. The greatest care should be taken to clean the patient at once, as soon as he has soiled himself.

The diet should be nutritious, but easily digestible. Tea, coffee, and alcoholics must be avoided.

Local antiphlogosis is important. This is best effected by means of Chapman's ice-bag (vide Fig. 33), or by an ordinary ice-bag or water-bag applied to the spine.

Derivatives may also be applied in the neighborhood of the spine, the best being alcoholic or other stimulating inunctions. But we object to the employment of dry cups, leeches, blisters, tartar emetic ointment, actual cautery, since they favor the development of decubitus. Applications of tincture of iodine to the spine must also be made with caution; inunctions of potassium iodide or iodoform (1 : 15) ointment or iodoform collodion are preferable.

Among internal remedies, potassium iodide (gr. xij., t. i. d.) is the most highly esteemed, but too much should not be expected from it. In anæmic individuals, preference should be given to the preparations of iodide of iron.

Patients who complain of painful twitchings in the limbs or other violent pains, require subcutaneous injections of morphine.

After the acute symptoms have subsided, we may employ the nervines, concerning whose efficacy opinions vary greatly. We may mention nitrate of silver, auro-natrium chloratum, strychnine, arsenic, phosphorus.

If irritative symptoms are absent, electrical treatment is much more strongly indicated at this stage of the disease. .

In electrical treatment of the site of. disease, the galvanic current is alone employed: current not too strong, sittings two to three times a week, not more than five minutes at a time. If there is only one focus of disease, the anode and cathode may be used stabile alternately, with large electrodes. Stabile ascending and descending currents may be employed alternately if there are several foci of disease.

Galvanization of the cervical sympathetic has also been recommended: one electrode on the sternum or last cervical vertebra, the other on the side of the neck immediately below the angle of the lower jaw .

The faradic current may be applied to the paralyzed muscles to prevent atrophy from disuse, and also in paralysis of the bladder and rectum.

After the patients begin to move their limbs, they must be warned against over-exerting themselves. They must especially avoid sexual intercourse.

Patients who are unable to travel may take baths during the summer, especially chloride of sodium or thermic baths. Cold-water cures some-

times produce surprisingly good results. In ordering baths of any
kind, it should be remembered that they should not be too warm (not
above 28° R.), too frequent (not more than three to four times a week),
or too prolonged (ten to fifteen minutes).

5. *Chronic Inflammation of the Spinal Cord.*

Chronic Myelitis.

I. ETIOLOGY.—The causes of chronic myelitis are the same as those
of the acute form, except that a congenital or acquired hereditary pre-
disposition is much more marked.

II. ANATOMICAL CHANGES.—The cord may appear normal to the
naked eye, although the microscope reveals extensive changes.

In many cases macroscopic changes are noticeable. As a rule, the
cord is unusually firm at the affected parts. These parts are generally
diminished in size, and the cord appears depressed and flattened in cir-
cumscribed localities. These have a gray or grayish-yellow color, appear
transparent, and sometimes shine through the pia mater. The meninges
over the affected parts are often thickened, adherent and congested, and
the pia mater can be removed with difficulty from the substance of the
cord.

In rarer cases, chronic myelitis appears as softening (myelomalacia).
After absorption of the inflammatory products, cavities may develop
(syringomyelia). The cavities, which are filled with serum, may attain
considerable size, or in other cases they impart to the cord a sieve-like
appearance (myelitis cribrosa).

Chronic myelitis affects the dorsal region most frequently, next the
cervical and lumbar enlargements.

The same conditions hold with regard to distribution and extent of
the inflammation as obtained in acute myelitis. If the gray matter is
chiefly or exclusively affected, the process is known as chronic central
myelitis.

Hallopeau has described a peri-ependymal sclerosis, which is confined,
in the main, to the connective tissue surrounding the central canal. In
chronic myelo-meningitis the peripheral portion of the white substance
is affected; this often follows a previous meningitis.

Chronic myelitis may also be circumscribed, transverse, and multiple
(disseminated, insular). The latter form must not be mistaken for sec-
ondary degeneration of the cord, which may follow a chronic inflamma-
tory process in the cord.

Among the microscopical changes in chronic myelitis, those in the interstitial
connective tissue are the most prominent, although a strict differentiation can-
not be made between interstitial and parenchymatous inflammatory processes.

The neuroglia presents increase of the interstitial tissue and the cellular ele-
ments. The spider cells are unusually abundant and distinct, often enlarged and
contain several nuclei. In addition, there are cells of a lower grade of develop-
ment, and even simple round cells. The interstitial tissue is gradually replaced by
distinctly fibrillated connective tissue. The blood-vessels are thickened, their nu-
clei increased, and their lymphatic sheaths unusually wide, and filled with fat,
granulo-fatty cells, pigment, or in places with white, more rarely red, blood-glob-
ules. The blood-vessels may be dilated in some parts, in others filled with clots.
Granulo-fatty cells may be entirely absent; at all events they are much rarer
than in acute myelitis. Amyloid bodies are more numerous.

The medullary sheaths of the nervous elements undergo degeneration. The
axis cylinders may present numerous spindle-shaped dilatations; the inflamma-

tory foci not infrequently contain numerous sclerosed, hypertrophic axis cylinders, without medullary sheaths. The ganglion cells are usually smaller, very granular and pigmented, and often retracted into small round clumps. In other places they contain vacuolæ.

Atrophic and sclerotic changes have been observed in the nerve roots and even in the peripheral nerves, in some cases. Degenerative atrophy of the muscles has also been seen.

Among other organic changes may be mentioned inflammation of the bladder, urinary passages and kidneys, pneumonic, phthisical, hypostatic changes in the lungs, decubitus, etc.

III. SYMPTOMS.—The symptoms begin not infrequently in an acute, febrile manner, or several acute relapses follow one another before the disease becomes chronic, while in other cases the development of the disease is slow and dragging from the start. Apart from the gradual onset of the affection, the symptoms are very similar to those of acute myelitis.

It often begins with paræsthesiæ, crawling, coldness, burning, etc. These are often associated with neuralgiform pains which may be very violent and are sometimes confined to definite localities, for example, the joints. Many patients complain of pains in the back and a cincture feeling. Paralyses of motion and sensation gradually supervene. The first signs of weakness generally appear first in the lower limbs, rarely in the upper limbs, somewhat more frequently in all the limbs. Spinal hemiplegia or even paralysis of a single limb may occur if the inflammatory focus in the cord is sufficiently circumscribed. The paralysis sometimes begins in the lower limbs, and then gradually extends to the upper limbs and even to the bulbar nerves. Paralysis of the bladder and rectum is sometimes an early symptom, and at all events it appears sooner or later. Priapism, seminal ejaculations, and impotence are also observed.

The same conditions obtain with regard to the reflexes, nutrition, and electrical excitability as in acute myelitis (vide page 85). In the later course of the disease we not infrequently notice tension and contracture of the paralyzed muscles and increase of the tendon reflexes—symptoms which are probably associated with secondary degeneration in the lateral columns of the cord.

Acute pyrexial exacerbations sometimes occur and are almost always followed by an aggravation of the symptoms, especially the paralysis. Erb states that he has seen the symptoms improve after typhoid fever and scarlatina.

The disease may last many years (ten to twenty); complete recovery hardly ever occurs, and some of the symptoms will persist even in the most favorable event. The disease sometimes takes an unexpected acute turn, the symptoms of acute myelitis develop, rapidly extend upwards, and prove fatal from paralysis of the bulbar nerves. Other patients die from gradual exhaustion, or bed-sores and cystitis develop, followed by pyæmic and septicæmic conditions. In some cases the clinical history changes and merges into that of multiple cerebro-spinal sclerosis.

IV.—DIAGNOSIS, PROGNOSIS, and TREATMENT are the same as in acute myelitis.

6. *Multiple Cerebro-spinal Sclerosis.*

I. ETIOLOGY.—The disease is most frequent between the fifteenth and thirty-fifth years, and is rare after the age of forty-five. According

FIG 35
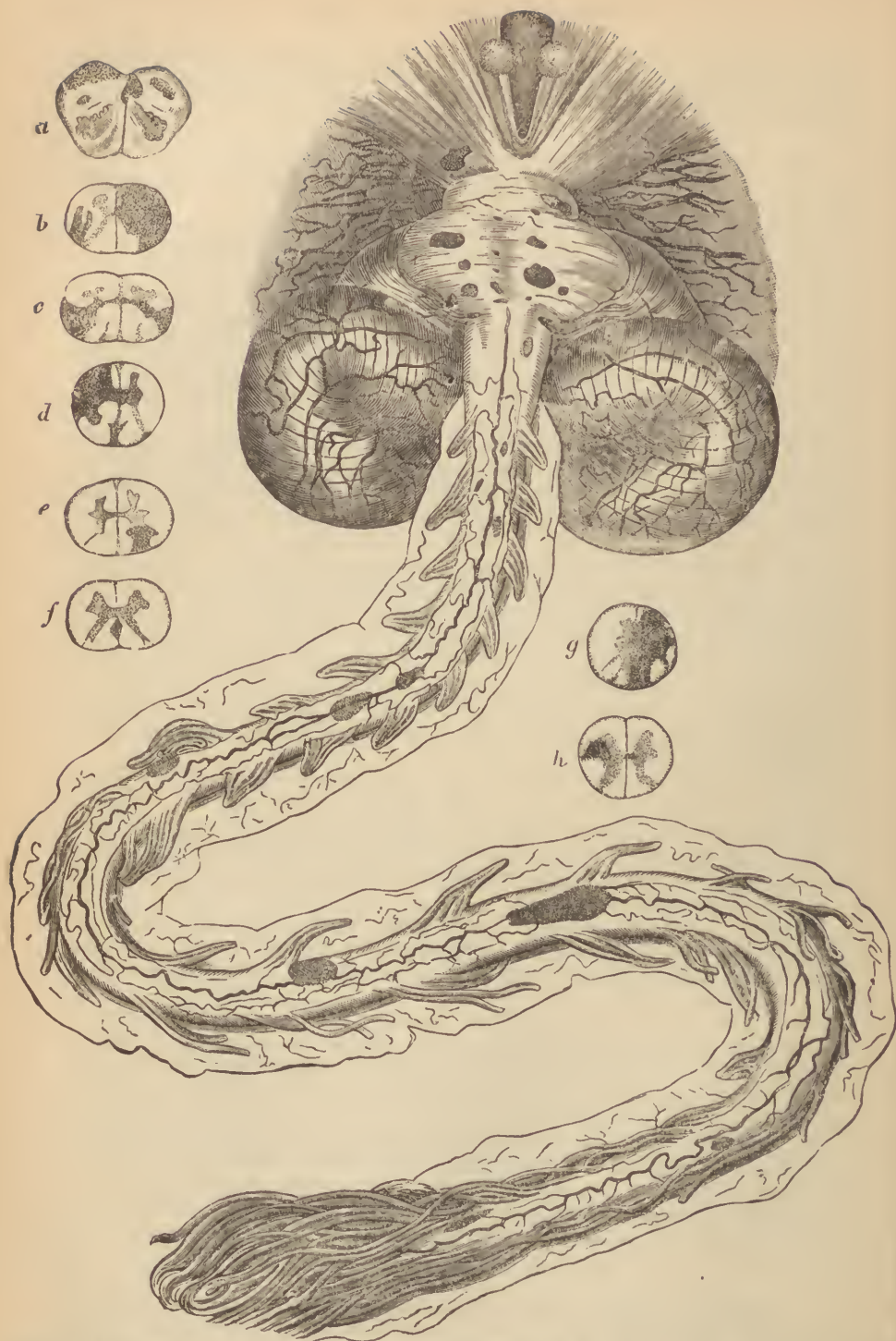
Spinal cord and posterior part of brain in multiple cerebro-spinal sclerosis. After Leyden. The sclerotic parts are readily recognized by their darker color. *a–h*, sections of the cord; *a*, medulla oblongata; *b, c*, cervical cord; *d, e*, dorsal cord; *f–h*, lumbar cord.

to Charcot and Moric it is not very infrequent in childhood. Three congenital cases (without autopsy) have been reported.

Heredity has been demonstrated in some cases. Many writers attribute the disease to cold, exposure to wet, injury or mental excitement. Pregnancy is said to favor its development. In some cases it follows acute infectious diseases, such as typhoid fever, variola, cholera, scarlatina, etc. According to some statements multiple sclerosis may be produced by hysteria (?).

II. ANATOMICAL CHANGES.—Multiple sclerosis appears in the form of inflammatory foci which are irregularly scattered throughout the brain and cord. Their number and size vary exceedingly. Sometimes there are more than a hundred foci, in other cases very few. Their size varies from a barely visible speck to a spot five to ten centimetres in size. Some foci are so small as to be visible only under the microscope.

FIG. 36.

Transverse section of the cervical enlargement in multiple sclerosis. Osmic acid preparation. The light parts in the spinal columns are sclerosed. Enlarged 10 times. After Bramwell.

The superficial plaques are generally visible through the pia mater with a grayish yellow, pearl-gray, or bluish-gray color. They sometimes project above the surrounding parts, sometimes they are depressed. As a rule, they are harder than the normal cord, more rarely they are gelatinous and soft. Transverse sections sometimes assume a light rosy-red color when exposed to the air.

As a rule, a more or less abundant juice can be scraped from them with the blade of the knife. The plaques vary in shape, and usually are sharply defined (vide Figs. 35 and 36). Under the microscope, however, they present gradual transitions into the normal tissues. The individual plaques are sometimes connected with each other by a less markedly inflamed portion, but this is not the rule, as is claimed by Buchwald. Many of the patches contain yellow dots and streaks (fatty, thickened vessels).

The inflammatory foci are distributed in an entirely irregular manner. In the cord the white substance is preferably affected; the changes are sometimes especially marked along certain columns. Secondary degenerations are generally absent, even if the sclerotic patches are so extensive as to interrupt the paths of conduction. Schulze attributes this to the fact that, on account of the long persistence of the axis cylinder, a real destruction of the conducting paths rarely occurs.

The medulla oblongata and pons are very often affected; the floor of the fourth ventricle presents a certain degree of predilection.

The cerebellum often escapes; if not, the sclerotic patches are almost always found in the white matter. This is also true of the cerebrum, in which the most frequently affected parts are the walls of the lateral ventricles, corpus callosum, centrum semiovale, corpus striatum, and optic thalamus.

In addition to multiple sclerosis, diffuse sclerosis of the brain has also been found.

The microscopical changes in multiple cerebro-spinal sclerosis are similar to those of chronic myelitis. The characteristic feature is proliferation of the connective tissue, with destruction of the nervous elements. In the most recent patches, Deiter's cells are found enlarged, and their nuclei increased; in addition, increase of the interstitial tissue. The neuroglia presents an unusual abundance of cells and nuclei, partly the result of increase of the elements previously present, partly of the emigration of white blood-globules from the vessels. Masses of round cells are found in many places on the external wall of the vessels. Finally, the interstitial tissue is converted into firm fibrillated connective tissue.

At the same time, the blood-vessels are thickened, are dilated into a spindle shape in places, their nuclei are increased; the lymphatic sheaths, which are dilated here and there, contain fat granules, granulo-fatty cells or larger accumulations of fat. On the external wall of the vessels we sometimes find accumulations of granulo-fatty cells, sometimes a larger or smaller number of fat crystals.

The number of granulo-fatty cells varies, but they disappear almost entirely in older plaques. The amyloid bodies are more numerous.

The medullary sheaths of the nerve fibres degenerate and disappear, the axis cylinders are dilated into a spindle shape, become firmer, and have a dull gloss. They are very resistant, and often persist in very old plaques. The ganglion cells often contain an unusual amount of yellow pigment, become granular, lose their prolongations, and are converted gradually into small round structures. The formation of vacuolæ within them is also observed.

Many authors believe that the lesion begins in the nervous elements proper, and that the proliferation of the neuroglia is a secondary process. Others regard the blood-vessels as the starting-point of the disease. It is not impossible that there are various modes of development. The blood-vessels have been regarded as the starting-point, particularly in those cases in which the process follows an infectious disease.

The spinal meninges are sometimes unchanged. At other times they are thickened, congested, and adherent over the sclerotic patches. Gray sclerotic patches have also been found in the cerebral nerves and the spinal nerve roots. Whether they also occur in the peripheral nerves has not been positively settled. Degenerative atrophy and fatty degeneration of the muscles have been observed at times, particularly when the anterior horns of gray matter have been affected. Leyden discovered sclerotic changes in the muscles.

Among the changes found in other organs may be mentioned: decubitus, cystitis, pyelonephritis, pneumonia, phthisis, bronchitis, and sometimes changes in the joints and bones.

III. SYMPTOMS.—The symptoms of multiple sclerosis present an unusually manifold character. Sometimes the cerebral, sometimes the

spinal symptoms predominate. But, as a rule, certain characteristic phenomena, viz.: volitional tremor, nystagmus, scanning speech, paretic symptoms, and apoplectiform attacks, are always observed.

At the beginning of the disease, the symptoms often have a vague character. Some patients complain of headache or vertigo, others of repeated attacks of vomiting and gastric pain (crises gastriques), still others manifest spinal disturbances, particularly paræsthesiæ and neuralgiform pains in the lower limbs.

The situation becomes more serious when paralytic symptoms develop. The motor disturbances are more prominent; sensory paralysis is sometimes absent during the entire course of the disease. The patients are easily tired in walking; the paresis gradually increases and extends to the trunk and upper limbs.

It is especially characteristic that each volitional movement is attended by violent tremor (volitional tremor). If the patient rises from a seat the trunk totters, generally from before backwards, and vice versa; when directed to move the head this part wags to and fro. If the patient is directed to place the fingers slowly upon the head of a pin, etc., the limb undergoes incessant zigzag, shaking movements. The more the patient's hand approaches the desired object the more vigorous the movements become. They cease, as a rule, during rest. This volitional tremor is so characteristic of multiple sclerosis that great, perhaps insurmountable obstacles will be encountered in diagnosis if the symptom is absent.

Charcot attributed the volitional tremor to the change in the axis cylinder of the sclerotic patches and to its persistence, so that the impulse of the will reaches the muscles in an intermittent manner. It is more probable that this symptom is the result of an affection of certain parts of the central nervous system, perhaps the pons and more anterior portions of the brain.

Nystagmus is an equally important symptom. It almost always develops when the patient is directed to look at an object steadily, or to follow with the eyes an object that is moved about. The eyes are then observed to twitch from one side to the other. Nystagmus is observed in about half the cases, but its cause is unknown.

There may also be very peculiar changes in speech. The patients speak slowly, scan the different syllables in a monotonous tone, often in a very high pitch. At a later period speech often becomes indistinct if paralysis and atrophy of the muscles of the tongue, palate, and lips become more marked. The pronunciation of the consonants l, p, g, and t is usually first affected.

Leube noticed, on laryngoscopic examination of a patient, that the closure of the vocal cords during phonation was suddenly interrupted for a short time; Lomikowski described trembling movements of the vocal cords.

Apoplectiform attacks occur in many cases during the course of the disease, sometimes at its onset, sometimes at a later period. The attacks generally begin suddenly, are attended with disturbance of consciousness, but rarely with complete unconsciousness, and sometimes with epileptiform convulsions. The bodily temperature is often markedly elevated (occasionally to 40° or 42°), and hemiplegia is often left over, but generally disappears in a few days. The entire condition is very often impaired after recovery from these attacks. In addition to the chief symptoms already discussed, there are a number of other phenomena of spinal, bulbar, or cerebral origin.

Sensory disturbances may be entirely absent. In other cases, certain qualities of sensation may be abolished, but complete anæsthesia is rare.

Paresis of the muscles is more frequent than complete paralysis. Rigidity and muscular spasms, which may develop spontaneously or only after purposive movements, are sometimes observed at a very early period. Contractures gradually develop, particularly in the muscles of the back of the neck and the adductors of the thigh, at a later period in the flexor muscles of the limbs, so that the patients become unable to walk. Previous to this the gait is awkward or stiff, like that of spastic spinal paralysis. Distinct ataxia occasionally develops if the posterior columns, particularly the bandelettes externes, are affected in a marked degree.

Paralysis of the bladder and rectum sometimes occurs at a very early period, but often disappears; at a later stage it remains persistent. This opens the way for the development of cystitis, pyelonephritis, ammonæmia, septicæmia, and pyæmia. The sexual function is gradually abolished.

Vaso-motor disturbances (abnormal color of the skin, changes in temperature and perspiration, gangrene and œdema of the skin) have been observed in some cases.

Trophic changes may also occur, viz.: decubitus, herpes, bullous eruptions, abnormal growth of hair, changes in the nails, swelling of the joints, etc. Individual muscles may rapidly atrophy and lose their faradic excitability; this takes place if the ganglion cells in the anterior horns of gray matter are destroyed. Increased galvanic excitability has sometimes been demonstrated in the contractured muscles.

Reflex excitability is markedly exaggerated in the majority of cases. This is true not alone of the cutaneous, but also of the tendon reflexes. Gentle tapping of the patellar tendon produces vigorous clonic contractions of the muscles of the corresponding limb, sometimes also in the other limb. Rapid dorsal flexion of the foot causes trembling movements of the whole limb, which often cease on sudden plantar flexion of the great toe (reflex inhibition). Vigorous reflex contractions are also produced by tapping the tendons of the biceps and triceps, etc.

If extensive changes occur in the medulla oblongata, symptoms develop which are similar to those of bulbar paralysis. The tongue undergoes atrophy and is moved with difficulty; speech becomes so indistinct as to terminate finally in a mere grunt; deglutition is impaired, closure of the epiglottis and of the naso-pharyngeal cavity is impaired, and the patients often swallow the wrong way; the lips atrophy, and can no longer be closed. Attacks of palpitation and dyspnœa occasionally set in, and may prove fatal (paralysis of the pneumogastric).

Other cerebral nerves may also be paralyzed, and this may give rise to deafness, unilateral or bilateral ageusia, anosmia, ptosis, paralysis of one or all the ocular muscles (in the latter event the eyeball is immovable), unilateral or bilateral myosis, or mydriasis. Many patients complain of flashes of light before the eyes, and gradually become amblyopic. Amaurosis is rare. Ophthalmoscopic examination reveals atrophy of the optic nerve, the papilla being abnormally white, and the retinal vessels unusually narrow.

Uhlhoff found atrophy of the disc in 25% of the cases, marked optic neuritis in 16%. Eulenburg recently described a case in which optic atrophy and complete amaurosis preceded the first spinal symptoms for five years.

Vertigo is a very frequent spinal symptom. The patients may have

the sensation either that objects are revolving around them or that they themselves are revolving. The vertigo is sometimes not of central origin, but the result of ocular paralysis and diplopia. There is often a remarkable variability of mood; the patients laugh and cry without motive. They often become apathetic and dull, and the facial expression becomes apathetic. Mania, dementia, delusions of grandeur, etc., develop in some cases.

The disease may last twenty or even thirty years. Remissions and exacerbations occur not infrequently. The former may be so pronounced as to raise the vain hope of recovery.

Charcot recognizes three stages of the disease: the first stage, until the development of contractures which compel the patient to keep constantly in bed; the second stage, in which the characteristic symptoms of the disease are present, and which often lasts many years; the third stage, a period of increasing marasmus.

The disease always terminates fatally. Death is the result of increasing marasmus, sometimes accelerated by violent diarrhœa; of decubitus and vesical paralysis, with their sequelæ; of an apoplectiform seizure; of advancing bulbar symptoms, or finally of intercurrent diseases.

IV. DIAGNOSIS.—In addition to nystagmus, disturbances of speech, vertigo, and apoplectiform attacks, volitional tremor possesses a very great diagnostic significance. The disease is not apt to be mistaken for other affections which are associated with tremor.

In *paralysis agitans*, the tremor persists during repose, hardly ever affects the head, and the patients are able to control it for a time by an effort of the will. In addition, the motor symptoms are less marked, sensory disturbances and paralysis of the bladder are absent, and the disease generally begins beyond the age of forty years.

In *chorea*, the movements present greater excursions and persist during repose; paralyses and sensory disturbances are absent.

Westphal recently described two cases in which the symptoms of multiple sclerosis existed for years, but the autopsy revealed no lesion of the nervous system. There is, therefore, a neurosis whose clinical history is so like that of multiple sclerosis that it cannot be distinguished from it with certainty. Optic nerve changes would be positive evidence in favor of the diagnosis of sclerosis.

On the other hand, there may be extensive multiple sclerosis without the typical clinical history of the disease. French writers have applied to this form the term sclérose en plaques fruste (atypical sclerosis). This variety sometimes presents only cerebral symptoms (vertigo, headache, apoplectiform or epileptiform attacks), although the autopsy shows extensive spinal sclerosis; other cases simulate the history of ordinary myelitis, etc.

The history of multiple sclerosis may also be very similar to that of locomotor ataxia. But in ataxia the patellar reflex is absent, and sensory symptoms predominate, while the motor power of the muscles is very little impaired. It may be very difficult to distinguish multiple sclerosis from progressive paralysis of the insane, since, apart from the fact that both affections may be combined, tremor, speech disturbances, pupillary changes and apoplectiform attacks may be observed in uncomplicated progressive paralysis.

V. PROGNOSIS.—The disease always terminates fatally, though life may be prolonged for a long time.

VI. TREATMENT.—The treatment is the same as that of myelitis (vide page 88).

7. *Tumors of the Substance of the Spinal Cord.*

I. ANATOMICAL CHANGES.—Tumors rarely occur in the substance of the spinal cord, and can hardly ever be diagnosed with certainty during life.

The most frequent form is glioma; next, pure sarcoma: myxosarcoma and fibrosarcoma have also been observed. Tubercles and gummata will be considered in Vol. IV.

Glioma affects mainly the enlargements of the cord, particularly the cervical enlargement. According to Virchow, it starts from the neuroglia; according to Klebs, from the nerve fibres and ganglion cells. It is sometimes unusually vascular (telangiectatic glioma), and is infiltrated with extravasations and blood cysts. Mixed forms of glioma are also observed.

The size of the tumors varies from that of a hempseed to that of a hazelnut, or even much larger. They are usually round, but occasionally elongated, and sometimes extend along the entire cord into the medulla oblongata. They are often sharply circumscribed, occasionally surrounded by a fibrous capsule ; more rarely they pass gradually into the substance of the cord.

The cord is often softened in the vicinity of the tumors, or secondary degeneration may result, etc.

II. SYMPTOMS.—Despite their large size, tumors of the cord sometimes remain entirely latent. In other cases, the most varied symptom complexes are observed. In sudden hemorrhages into and rapid enlargement of a tumor, the symptoms of hæmatomyelia are produced. More or less acute signs of compression of the cord are most apt to develop. Or the signs of transverse ascending and descending myelitis are produced. Tabetic or spastic symptoms are noticed, according as the posterior or lateral columns are involved. The symptoms of progressive muscular atrophy or the various forms of poliomyelitis have been observed, if the ganglion cells of the anterior horns were affected. The symptoms of unilateral lesion of the cord have also been described. Or vague, indefinable spinal symptoms make their appearance. The disease may last for many years. Death results from intercurrent diseases or increasing marasmus.

III. ETIOLOGY.—Nothing is known concerning the etiology of the tumors. Trauma, cold, pregnancy, mental excitement have been mentioned as causes.

IV. DIAGNOSIS.—The diagnosis is generally impossible. As a rule, we must be satisfied with the diagnosis of a spinal affection. We may suspect a neoplasm if the symptoms have been preceded by phthisis, scrofula, or syphilis.

V. PROGNOSIS.—With the exception of gummata, the prognosis is always bad.

VI. TREATMENT.—Gummata are treated by antisyphilitic remedies. The treatment of other forms of tumor is purely symptomatic.

8. *Syringomyelia and Hydromyelia.   Formation of Cavities in the Spinal Cord.*

1. Cavities in the cord may be congenital or acquired. They sometimes extend the entire length of the cord, from the lower part of the fourth ventricle to the conus terminalis, somtimes they are very short. On transverse section, they form narrow fissures or elongated, rounded, or irregular cavities; their size often varies in different places. A number of cavities are sometimes closely aggregated. They are generally most extensive in the cervical and dorsal portions of the cord, so that these parts may form a fluctuating sac, which generally contains clear serous fluid. The fluid is rarely hemorrhagic or flocculent.

The wall of the cavity is often composed of firm connective tissue. Under the microscope, this is found to consist of branching cells, fibrillated intercellular substance, a few granulo-fatty cells, and thickened blood-vessels. The latter may be very brittle, and give rise to hemorrhages. In other cases, the cavity is situated in a gray, gelatinous mass composed of intercellular substance and round cells. The inner surface of the cavity sometimes is covered with cylindrical epithelium; sometimes this is absent.

The cavities are situated most frequently between the posterior columns of the cord, but often change their position at different heights. They are often independent of the central canal; in other cases they merge into it in places.

Sclerosis, especially of the columns of Goll or Burdach, acute and chronic mye-
litic foci, etc., are also often present.

2. Simon suggested that a distinction should be made between syringomyelia
and hydromyelia, the latter term being applied to those cavities which result
from dilatation of the central canal.   These dilatations, apart from congenital
cavities, may be the result of inflammatory proliferation and subsequent retrac-
tion of the connective tissue surrounding the central canal or of circulatory dis-
turbances in the cerebellar fossa as the result of tumors.

Simon and Westphal believe that softening of tumors and subsequent absorp-
tion may give rise to the formation of cavities in the cord, but Leyden claims
that these are apt to be mistaken for inflammatory new-formations or coagulation
at the periphery of the cavities.

Hemorrhages and inflammatory softening may also lead to the formation of
cavities, after absorption has taken place.   Simon attributes some cases to men-
ingeal adhesions.

FIG. 37.   FIG. 38.
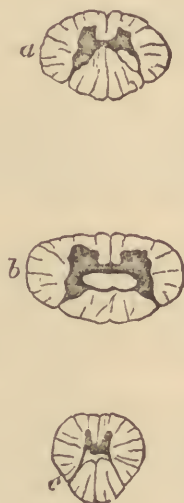
Two cases of syringomyelia without symptoms during life.   a–b, cervical cord ; c–e, dorsal cord.
Natural size.

Eichhorst and Naunyn observed syringomyelia develop in new-born dogs,
whose spinal cord had been strongly compressed at one spot soon after birth.

3. In a number of cases, very large cavities have been found which had given
rise to no symptoms during life, as is shown in two cases which came under our
own observation (Figs. 37 and 38).   Spinal symptoms were absent in both cases,
although the cavities extended from the first cervical to the first lumbar nerves.
An attempt has been made recently to construct a symptom complex for certain
cases: slowly-progressing atrophy of the upper limbs, partial sensory disturb-
ances, especially changes in temperature and pain sensibility, vaso-motor and
trophic changes, absence of the tendon reflexes, rarely rigidity of the muscles
and contractures; bulbar symptoms often supervene at a later period.

b. SYSTEM DISEASES OF THE SPINAL CORD.

SIMPLE SYSTEM DISEASES.

1. *Gray Degeneration of the Posterior Columns.   Tabes dorsalis.*

(Sclerosis of the Posterior Columns.   Progressive locomotor ataxia.)

I. ETIOLOGY.—Locomotor ataxia is the most frequent disease of the spinal cord. It is much more common among men than among women, and is chiefly a disease of middle life, particularly between the ages of 30 and 40 years. It rarely begins after the age of 50 years or during the period of childhood.

Among its causes syphilis occupies a prominent part. Although our experience corroborates that of Leyden and Westphal, who maintain that Fournier and Erb have exaggerated the importance of this etiological factor, nevertheless it cannot be denied that a history of syphilis is often obtained in this disease, and that an inunction treatment produces surprising results in not a few cases.

From our present knowledge it would appear that ataxia is particularly apt to follow syphilis if the symptoms of the latter were mild and were not thoroughly treated. Tabes dorsalis is one of the late sequelæ of syphilis (usually five years, often ten to twenty years, after infection). Some writers maintain that syphilis merely causes a sort of weakness of the spinal cord, so that other hardly demonstrable causes may give rise to tabes.

Cold is often mentioned as a cause, but it is probable that this will act as an etiological factor only if there is an abnormally slight power of resistance of the spinal cord, either congenital or acquired. Among causes of a similar character may be mentioned: over-exertion, injury, concussion of the spine, mental excitement, excessive lactation, sexual excesses, etc.

The disease sometimes appears after acute infectious diseases, such as pneumonia, typhoid fever, articular rheumatism, cholera, etc.

Tuczek observed the symptoms of ataxia after chronic poisoning with ergot, and Leyden makes a similar statement with regard to pellagra.

Heredity plays a prominent part in certain cases. Thus, Carré observed the disease in the grandmother, mother, seven brothers and sisters, and nine other relatives.

Westphal showed that the disease develops not infrequently during the course of progressive paralysis of the insane.

II. ANATOMICAL CHANGES.—In exceptional cases the spinal cord appears normal to the naked eye, although the microscope reveals advanced changes.

The disease is often evident from the shape, color, and consistence of the cord. The posterior surface appears flattened and narrowed, and if the pia mater has remained transparent, a gray, pearl-gray, or yellowish-gray tissue is seen in a position corresponding to the posterior columns. As a rule, the consistence of this part is increased, rarely diminished. These appearances are often concealed by changes in the pia mater, which is thickened and opaque over the affected parts of the cord, contains numerous sinuous vessels, is occasionally markedly pigmented and adherent to the arachnoid and dura mater. Opacities and calcareous plates are often found on the arachnoid and dura mater.

On transverse section of the cord, the posterior columns are found to have a pearl-gray or yellowish-gray color. These parts are often transparent; they are sometimes unusually firm, in other cases soft and gelatinous. The changes are generally most marked in the upper lumbar and lower dorsal portions of the cord (vide Fig. 39). In the lower lumbar region they are often confined to the columns of Burdach, in the cervical region to the columns of Goll. Even where the changes are most marked, islets of the posterior columns often remain intact. This is especially true, as is shown in Fig. 40, of the vicinity of the posterior gray commissure and the part immediately adjacent to the posterior horns. The changes may sometimes be followed to the medulla oblongata into the funiculi graciles, and gradually disappear. In a few cases, changes have also been found in the peripheral layers of the pons and the corpora quadrigemina.

The posterior spinal roots are atrophic and gray, and present changes similar to those observed in the cord. These changes never extend beyond the intervertebral ganglia. The cauda equina contains thin, gray strands which correspond exactly to the atrophic posterior spinal roots.

Gray degeneration of the cerebral nerves has been observed in a number of cases, particularly in the optic nerve. It begins in the immediate vicinity of the eyeball and the peripheral layers of the trunk of the nerve, and may gradually extend along the optic tract to the geniculate bodies. Similar changes have been observed in some of the nerves of the ocular muscles, in the trigeminus, hypoglossus and pneumogastric, even in the nuclei on the floor of the fourth ventricle.

The brain presents no change, unless the ataxia is a complication of progressive paralysis of the insane.

Friedreich first noticed atrophy of nerve fibres, and increase of the interstitial tissue in the peripheral nerves. Degenerative and atrophic conditions have also been observed in the nerves of the muscles.

FIG. 39.

Transverse sections of the cord in advanced degeneration of the posterior columns. a, upper cervical region; b, cervical enlargement; c, upper; d, middle; e, lower dorsal cord; f, lumbar enlargement. The sclerotic portions are light in color. Natural size. After Leyden.

Microscopical examination of the degenerated portions of the posterior columns shows disappearance of the nerve fibres, and the presence either of a rather large-meshed connective tissue, which is poor in cells (vide Fig. 41) or of a more delicately fibrillated connective tissue (vide Fig. 42). In some parts the connective tissue is firmly fibrillated.

Granulo-fatty cells are entirely absent in places, in others they are more or

less numerous. The number of amyloid bodies is very considerable. The blood vessels are usually thickened, and their walls contain an unusual number of nuclei. Their lymph sheaths may contain accumulations of fat granules and gran-

FIG. 40.

Transverse section of the middle dorsal region. The degenerated parts are light in color. Enlarged 10 times. After Leyden.

ulo-fatty cells. The nerve fibres are partly destroyed, in part they consist of naked axis cylinders, the medullary sheath having undergone absorption.

Opinions differ with regard to the nature of these changes. Leyden looks upon the process as a primary, non-inflammatory degeneration of the nerve

FIG. 41.   FIG. 42.

From the degenerated foci in the cord.
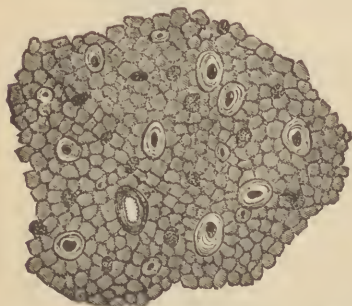
Fig. 41.—Tissue with coarse and firm network.   Fig. 42.—With finer network. Contains thickened blood-vessels and persistent nerve fibres. After Leyden.
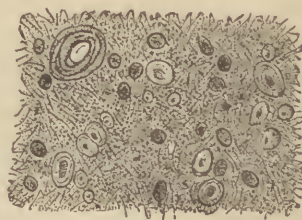
fibres, with secondary implication of the connective tissue. Others regard the process as primarily inflammatory; Charcot regards the nervous tissue as the starting-point of the inflammation (chronic parenchymatous myelitis) and the

connective-tissue changes as secondary, others consider the process a chronic primary interstitial myelitis.

Some observers have associated the disease with primary changes in the blood-
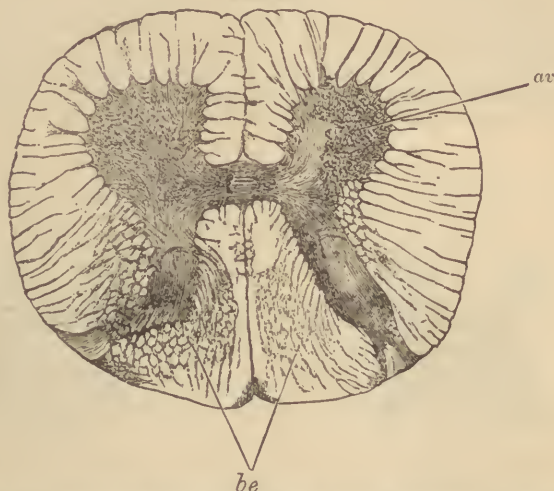
Transverse section of the cervical enlargement in tabes, with atrophy of right anterior horn. After Charcot. *av*, atrophic anterior horn; *be*, bandelettes externes.

vessels. Still other writers regard the inflammation of the pia mater and arachnoid as the primary lesion.

The posterior horns of gray matter usually take part in the process. They ap-

FIG. 44.   FIG. 45.
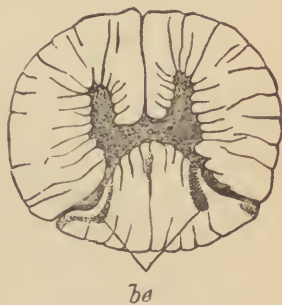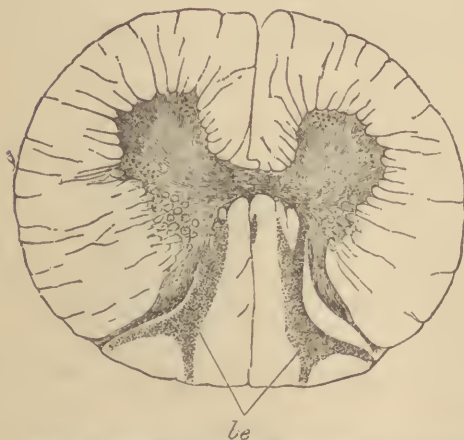
Gray degeneration of the bandelettes externes in beginning tabes. After Charcot.
Fig. 44.—Middle cervical region.
Fig. 45.—Lower dorsal region; *be*, bandelettes externes.

pear small, their nerve fibres are diminished in size or have disappeared, the connective tissue is unusually abundant, and the ganglion cells are shrunken, often filled with an unusual amount of yellow pigment, and many of the cells are destroyed.

Clarke's columns in the dorsal cord often present a grayish discoloration to the naked eye.   Under the microscope we find atrophy and disappearance of the nerve fibres and increase of the connective tissue, but the ganglion cells are usually intact.

The morbid process often extends from the posterior columns along the periphery to the lateral columns.   Sometimes it extends only to the cerebellar tracts, in other cases it passes deeper into the lateral columns, occasionally it even extends to the anterior columns.   Finally, Charcot and Pierret found that, in rare cases, the morbid process extends to the large ganglion cells of the anterior horns.   After undergoing pigment degeneration, the ganglion cells shrink and disappear, and the corresponding anterior horn appears atrophic (vide Fig. 43).   These

Fig. 46.
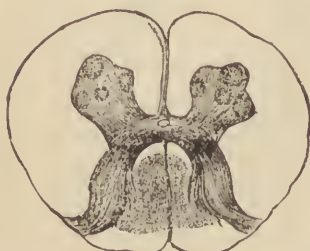
Beginning changes in lumbar cord in tabes.
After Struempell.

Fig. 47.

Advanced changes in lumbar cord in tabes.
After Struempell.

processes correspond to atrophy of muscles, and degeneration reaction during life.

Very little is known concerning the post-mortem appearances in the initial stages of tabes.   According to Charcot, the primary site of disease is situated in the internal root fibres (bandelettes externes) in the lateral part of the column of Burdach near the posterior horn.   All the other anatomical changes are said to be secondary in character, particularly the affection of the columns of Goll, which is regarded merely as an ascending secondary degeneration.

Struempell has come to the following conclusions with regard to the distribution of the lesions:

In the lumbar cord, the changes begin symmetrically in the middle portions

Fig. 48.

Beginning changes in dorsal cord in tabes.
After Struempell.

Fig. 49.
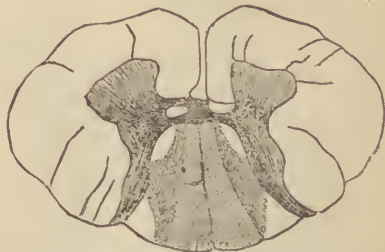
Changes in cervical cord in tabes.   After Struempell.

of the posterior columns, and intact parts are left anteriorly between them and the posterior gray commissure and posteriorly between them and the outer boundary of the posterior columns (vide Fig. 46).   As the disease progresses, the process extends posteriorly, but a small oval or triangular field, along the posterior longitudinal sulcus, is generally left intact (vide Fig. 47).   The most anterior portions of the posterior columns always remain intact.

In the dorsal cord, the process begins in two lateral fields from which fibres mainly radiate into the posterior horns.   In addition, two median strips, which

are situated along the posterior longitudinal sulcus, are affected very early (vide Fig. 48). Later, the entire posterior column degenerates, but the posterior external portion persists unchanged for an unusual length of time.

In the cervical cord, the change is first noticed in the columns of Goll, more markedly posteriorly than anteriorly. Two anterior and two posterior external fields remain intact for a long time (vide Fig. 49).

It would appear, accordingly, as if the total degeneration of the posterior columns consists of the gradual affection of smaller systems of fibres which are functionally similar.

In some cases, scattered sclerotic patches have also been found throughout the cord.

III. SYMPTOMS.—The most important and constant symptom of tabes dorsalis is inco-ordination of voluntary movements, ataxia, with unimpaired or slightly diminished muscular power.

Locomotor ataxia is an exquisitely chronic disease, which may last more than thirty years.

Leyden distinguishes a neuralgic, ataxic, and paraplegic stage.

In the first (neuralgic) stage neuralgiform pains play a prominent part. These may be associated with paræsthesiæ, ocular paralyses, pupillary changes, optic atrophy and amaurosis, loss of the patellar tendon reflex, anæsthesiæ, disturbances of the bladder and rectum, and changes in the joints. This stage varies in duration from several weeks to many years.

In the second (ataxic) stage, the symptoms of ataxia become more prominent. These almost always appear first in the lower limbs, often at a later period in the arms; they rarely begin in the upper limbs. This stage may also last many years.

The third (paraplegic) stage begins when the patient is no longer able to move the lower limbs, and is confined to bed. In some cases, death is the result of bed-sores, paralysis of the bladder with alkaline decomposition of the urine, ulceration and necrosis of the vesical mucous membrane or pyelonephritis. Others die of pulmonary phthisis (a frequent termination), or general marasmus, which is not infrequently accelerated by profuse diarrhœa or by obstinate vomiting as the result of so-called gastric crises, or intercurrent diseases. In several cases, Leyden observed acute delirium with coma, and a fatal termination.

We will now consider the individual symptoms somewhat more in detail.

Neuralgiform pains are among the most constant and earliest symptoms. As a rule, they are described as lightning-like, shooting, sticking, boring, or crushing. They are often so violent that the patients cry out aloud. They are almost always situated deeply, rarely in the integument; the latter is often hyperæsthetic in the situation of the pains. The pains sometimes appear along certain nerve tracts, sometimes as hemicrania, or chiefly in the joints, or they may not follow a sharply defined anatomical course. They are often especially severe at night. Remissions and exacerbations are frequent, according to the temperature and barometer; the exacerbations are most marked in rainy, stormy weather, with a low barometer.

As a rule, the pains first appear in one, then in the other lower limb, and are not infrequently regarded as sciatica. The pains usually appear in the upper limbs at a later period, and are apt to extend along the distribution of the ulnar nerve.

The majority of the patients also complain of girdling pains (cincture feeling) around the chest or abdomen.

Visceral neuralgias may also develop; violent gastralgia with vomiting (gastric crises), violent burning in the urethra and rectum, associated with dysuria and tenesmus, occasionally with dysentery-form stools, etc.

In rare cases, the pains are felt along the cerebral nerves, generally along the occipital, next the trigeminal nerves.

The pains often continue during the further course of the disease.

Paræsthesiæ constitute a very constant symptom, and, as a rule, develop at a very early period. The patients complain of a feeling of coldness, burning, prickling, or formication. Many complain that they do not feel the floor in walking, that they have a sensation as if walking on felt, wool, or feathers, or on a bladder filled with water. The paræsthesiæ usually appear first in the lower limbs, and are generally more marked in the feet and legs. At a later period, they extend to the upper limbs, and even the trunk may be affected.

Hyperæsthesia is not a very frequent symptom. It is sometimes associated with the neuralgiform pains in the skin, and may rapidly change its location.

Some patients do not experience pain on irritation of the skin unless the irritant is very severe, but the pain is then unusually violent. Leyden has applied to this phenomenon the term relative hyperæsthesia.

The patients rarely complain of pain in the back. M. Meyer has called attention to the presence of pressure points on the spine, and has had good results from treating them with electricity.

According to Leyden, anæsthesia occurs in every case of tabes, but careful examination is sometimes requisite in order to detect it. In many cases there is partial anæsthesia, in more advanced cases it may be complete. It is generally most marked in the feet and legs, but in the beginning of the disease it is sometimes confined to circumscribed parts of the integument, for example, the perineum, the lateral and posterior part of the thigh, scrotum, etc. The sensation of tickling is generally lost first, then the sensation to touch and pain, later the other modes of sensation.

Delayed conduction is a not infrequent symptom. If the skin is pricked with a pin, several seconds (up to five) may elapse before the sensation is perceived by the patient. Hertzberg and Nothnagel also observed delayed conduction of temperature impressions.

Naunyn recently described double sensation, i. e., the patient, after a single prick of a pin, first experienced pain, and after an interval, a second, more intense pain. E. Remak noticed in one patient that the prick of a pin was first felt as a simple touch, and after an interval as pain.

Fischer noticed polyæsthesia, i. e., one point was felt as several. In the early stages of the disease, Berger noticed that feeble cutaneous irritants were perceived, but strong ones were not perceived. E. Remak calls attention to the readiness with which the sensory nerves are exhausted. If the skin is faradized, the patient, after a while, states that he no longer feels anything. The current must then be increased, but again a point arrives at which the sensation which was previously present disappears. Mendelssohn showed that the electrical reaction of the sensory cutaneous nerves is changed in tabes. There is often a prolonged after-sensation, so that rapidly following stimuli merge into one.

Leyden's investigations have shown that the anæsthesia affects not alone the skin, but also the fasciæ, tendons, and muscles. The impairment of muscular sense is especially important.

If the patient's leg is slowly and carefully raised while his eyes are closed, and he is directed to raise the other leg to the same extent, gross mistakes are made. If the leg is placed in a sling, and passive movements of the thigh, leg, or foot are made, the patient is unable to tell the position of the limb. If one leg is carefully placed over the other he is unable to tell which leg is uppermost. If he is directed, while the eyes are closed, to bring the fingers of the two hands in contact, he hunts for them in the air, etc.

The sense of effort, however, is retained, according to Leyden. This is tested by placing a folded handkerchief upon the extended limb, after the latter has been placed upon a support, and directing the patient, with the eyes closed, to tell differences in weights placed upon the handkerchief. Electro-muscular sensibility may also be unchanged, but it is sometimes diminished.

The Brach-Romberg symptom is generally attributed to changes in the muscular sense. This is shown by the tottering movements of the patient in the dark, or when the eyes are closed. It is evident that in the light the eye is able, to a certain extent, to overcome the defect in the muscular sense. The tottering is especially marked when the feet are brought together.

The cutaneous reflexes present no characteristic changes. They may be unchanged, diminished, increased, or delayed. When the prick of a pin on the sole of the foot gives rise to reflex contractions of the leg, the patient sometimes mistakes the perception of the muscular contractions for that of the prick itself.

The condition of the patellar tendon reflex is an extremely important symptom. This is tested by directing the patient to cross one knee over the other, and by tapping the tendon above the patella quickly with the percussion hammer or the ulnar side of the hand. Or the patient allows the limb to hang loosely over the edge of the bed, and the patella tendon is then tapped. Or the physician's hand is placed under the knee-joint of the patient, the limb is slightly elevated, and the tendon percussed. In tabetic patients, the reflex (contraction of the quadriceps and propulsion of the leg) remains absent in almost every case. This is one of the earliest symptoms of ataxia. Its appearance indicates the existence of foci of degeneration in the external portions of the posterior columns in the upper lumbar region or its transition to the dorsal region.

Vaso-motor, secretory, and trophic changes may remain absent during the entire course of the disease. In other cases there is a diminution in the temperature of the skin of the affected limbs corresponding to a sensation of coldness. Unilateral and local hyperidrosis and ptyalism have been observed. Erythema, herpes, bullous and nodular eruptions may appear, and acute decubitus with its sequelæ may also develop. Pooley described unusual desquamation of the epidermis; Ballet and Dutil observed ichthyotic changes on parts of the skin which were anæsthetic, hyperæsthetic, or the site of severe pains. Strauss described cutaneous hemorrhages. Thickening, fissuring, and falling out of the nails have been reported in a few cases. Falling out of the teeth has also been reported, and according to Mayeraudière, this is always preceded by hyperæsthesia or anæsthesia at the site of the tooth.

The panniculus adiposus and muscles may remain normal for a long time, and emaciation does not occur, in many cases, until the patient has been confined to bed for a long time. But rapid atrophy and degeneration reaction (usually in the upper limbs) occur if the ganglion cells of the anterior horns are destroyed. According to Charcot, an important, early diagnostic sign of tabes is hemiatrophy of the tongue, generally associated with ocular paralysis.

Charcot also includes among the trophic disturbances certain peculiar joint changes, which often occur very early, more rarely in the later stages of the disease.

The knee-joint is more frequently affected, next the shoulder (especially on the right side), elbow, hip, and wrist. The disease is unattended with fever or pain, but with marked swelling which sometimes extends over a considerable part of the limb. The swelling, which generally consists of an accumulation of serum or sero-bloody fluid in the joint, may disappear after weeks or months without evil effects, or it terminates in erosion of the cartilages and ends of the bones; the joints then creak on motion, and spontaneous dislocations may develop (vide Fig. 50). Suppuration and perforation of the joint are very rarely observed.

Charcot has also noticed fragility of the bones, terminating in spontaneous fractures. The lower limbs are generally affected, especially the thighs. Recovery is not infrequently attended with an abnormal development of callus. In such cases, Blanchard found disappearance of the lime salts and rarefaction of the osseous tissues in the neighborhoood of the Haversian canals, most markedly near the medullary cavity.

These changes are connected with the development of the tabetic foot. In

FIG. 50.   FIG. 51.

Fig. 50.—Eroded head of humerus in tabes dorsalis.

Fig. 51.—Normal head of humerus. After Charcot.

this condition the scaphoid and internal cuneiform bones project strongly on the inside, while the metatarsus falls back on the outside, so that a characteristic projection forms at the first tarso-metatarsal joint.

In one case Kroenig described spondylolisthesis. Among other secretory and trophic disturbances we may finally mention varicocele, swelling of the testicle, and mal perforant du pied.

Motor disturbances play a prominent part in the symptomatology of tabes. Paralysis is either altogether absent or occurs only at a late stage of the disease as the simple result of prolonged disuse. Temporary hemiplegic or paraplegic paralyses are observed occasionally, and are regarded by Leyden as exhaustion paralyses. But despite the fact that the patients generally possess sufficient power, they present the signs of motor inco-ordination or ataxia. In walking, the legs are separated widely, the heels are brought forcibly on the ground, and irregular movements are performed in lifting the limbs. The gait becomes uncertain and zigzag, and the patients are unable to walk in a straight line. These disturbances become especially marked on making sudden turns, walk-

ing up-stairs, etc., or on closing the eyes. The patient carefully follows every movement with his eyes. As the symptom progresses, he is no longer able to walk without assistance. Finally, the feet make such violent, irregular movements that he is confined permanently to the chair or bed. Ataxic movements sometimes occur in the upper limbs, so that the patient performs irregular, unnecessary movements in threading a needle, etc. In advanced stages he is unable to write, eat, or clothe himself, and becomes as helpless as a child. The electro-muscular irritability remains unchanged.

According to Leyden, ataxia is merely the result of extensive anæsthesia (sensory ataxia), including not alone cutaneous anæsthesia, but particularly diminution or abolition of the muscular sense. It has been contended, in opposition to this view, that ataxia may exist without disturbance of sensation, and also that anæsthesia occurs without ataxia. Friedreich explains ataxia by the paralysis of definite co-ordinating nerve tracts (motor ataxia) which convey the co-ordinating impulse from the centre in the brain through the posterior columns (or cerebellar tracts?) to the periphery. It must be remembered, however, that no nerve tracts of co-ordination have yet been discovered. Cyon believes that ataxia is the result of an interruption of reflex paths in the spinal cord.

Muscular twitchings are rare and generally result from neuralgic pains; contracture of the muscles is equally rare.

Disturbances of the bladder and rectum are frequent and often very early symptoms. The patients often suffer from incontinence or from inability to empty the bladder. In the later stages of the disease, complete paralysis of the bladder may occur and result in stasis of urine, cystitis, pyelitis, nephritis, pyæmia, and septicæmia. There may also be involuntary evacuations from the bowels, or obstinate constipation.

Ocular symptoms are very often present and are extremely important in diagnosis. Paralysis of ocular muscles is shown by ptosis, strabismus, diplopia, and vertigo. The motor oculi is most frequently affected, then the abducens, and then the trochlearis.

The pupils are often no larger than the head of a pin (spinal myosis). They react very little or not at all to light, but change their dimensions during accommodation. The pupils are sometimes unequal.

Amblyopia is often a very early symptom, and may terminate in complete blindness. This is sometimes observed for years prior to the first distinct signs of ataxia. The ophthalmoscope shows a very white color of the disc, the lamina cribrosa is unusually distinct, the retinal arteries narrowed.

The field of vision is more and more constricted, not concentrically, but by the loss of sectors. There are also peculiar disturbances of color sense, viz., the development of insensibility to certain colors; first for red and green; yellow, and particularly blue, are retained longest.

Visceral crises is the term applied to peculiar attacks which affect one or more of the viscera (stomach, larynx, pharynx, kidneys, urethra, etc.).

Gastric crises are the best known among these symptoms. They consist of attacks of violent gastric pain, associated with protracted gagging and vomiting, at first of the gastric contents, later of mucus, and then bilious fluid. These attacks sometimes last only a few hours, sometimes several days. They produce great exhaustion, and even

terminate fatally. Such conditions sometimes follow or are associated with unusually violent neuralgiform pains. Diarrhœa and hemorrhages from the genitals (in women) have been observed during the attacks. James Russel noticed diminution of urine and urea, and albuminuria. Buzzard states that gastric crises occur particularly in patients who suffer from joint affections. They are sometimes an early symptom (prior to the ataxia), but recur from time to time at a later period.

Lépine also describes painless gastric crises, characterized by profuse vomiting, which may be so obstinate as to endanger life.

Intestinal crises consist of attacks of diarrhœa associated with pain ; they may be attended by salivation and hyperidrosis.

Laryngeal and pharyngeal crises are attacks of dyspnœa and suffocation and difficulty in deglutition, which may be associated with cyanosis and temporary protrusio bulbi.

In renal crises there are violent attacks of pain, similar to renal colic; the secretion of urine is diminished, but it contains no blood or pus. The attacks may last a week.

Pitres has observed, in women, clitoris crises, i. e., attacks of voluptous sensations similar to those experienced during coitus. Priapistic and spermatorrhœic crises have been observed in men.

The development of the crises has been attributed in part to degeneration of the peripheral nerves, in part to degeneration of the nuclei of the cerebral nerves.

Circulatory symptoms have been described in a number of cases. The pulse is often very rapid, and attacks of palpitation, terror, and pain in the heart (crises cardiaques) are also mentioned. Eulenburg noticed very marked dicrotism of the pulse. Berger and Rosenbach noted the frequent coincidence of tabes and aortic insufficiency.

Diseases of the cerebral nerves, apart from the optic and the nerves distributed to the ocular muscles, occur quite rarely. Tinnitus aurium and hardness of hearing have been described in a number of cases. Trigeminus anæsthesia is sometimes observed ; likewise paralysis and spasms in the distribution of the facial nerve.

Disturbances of the sexual functions are observed particularly in males. At the onset of the disease there is often increased sexual desire, and the semen is discharged very quickly ; pollutions occur very often. These symptoms, however, gradually give way to impotence.

The psychical functions are often affected. The majority of writers mention the cheerful disposition of the patients; but, if closely watched, this very disposition is often seen to be morbid. Well-marked insanity occurred seventeen times in eighty-nine tabetic patients (Mœli). Bernhardt recently called attention to apoplectiform attacks, but these occur much less frequently in tabes than in multiple sclerosis.

The following tables by Bernhardt (58 cases) and Erb (56 cases) show the frequency of the individual symptoms in this disease :

	Bernhardt.		Erb.	
1. Absence of patellar tendon reflex.........	100.0	per cent	98.0	per cent.
2. Lancinating pains......................	79.5	"	92.5	"
3. Paresis of bladder......................	74.1	"	81.0	"
4. Paralysis of ocular muscles.............	39.6	"	38.7	"
5. Myosis..............................	27.2	"	54.0	"
6. Rigidity of pupils......................	48.4	"	50.0	"
7. Optic atrophy	10.3	"	12.4	"
8. Sensory disturbances..................	85.9	"		
9. Analgesia	31.6	"	69.0	"
10. Delayed conduction of pain..............	34.4	"	89.5	"
11. Brach-Romberg symptom................	90.2	"	83.5	"
12. Feeling of exhaustion.......	92.0	"	97.9	"
13. Ataxia.............................	94.1	"	100.0	"
14. Sexual weakness......................	43.7	"	78.5	"
15. Joint affection	9.6	"		
16. Crises gastriques.....................	5.2	"		

IV. DIAGNOSIS.—At the onset of the disease it may be mistaken for rheumatism, hemicrania, sciatica, gastric catarrh, hypochondria, and hysteria. We must take into consideration the condition of the patellar reflex, the reflex rigidity of the pupil, temporary ocular paralyses, myosis, irregularity of the pupils, sensory disturbances, paralysis of the bladder and rectum. The earliest symptoms, and one of the most important, is the absence of the patellar tendon reflex. This is occasionally absent in healthy individuals. It may be absent in individuals who belong to neuropathic families, in old age, and, according to Schreiber, in chronic alcoholism.

With regard to the diagnostic significance of ataxia, it must be remembered that it may occur in diseases of the cerebellum (cerebellar ataxia), and that it also appears occasionally after acute infectious diseases (acute ataxia). In the former event other cerebellar symptoms will be present, such as constant vomiting, occipital pain, vertigo, etc.

Déjérine has recently claimed that degenerative changes in the peripheral nerves alone may produce the symptomatology of tabes dorsalis. He applies to the disease the term neurotabes périphérique.

V. PROGNOSIS.—The prognosis is always unfavorable. The hope of improvement, rarely of complete recovery, can only be entertained if the disease is the result of syphilis.

VI. TREATMENT.—If there is a suspicion of syphilis, we should order mercurial inunctions, and give potassium iodide internally. The symptoms may grow worse for a few weeks after the inunctions are begun, but their continued application sometimes produces remarkable results.

If the patient has not had syphilis, nothing remains except to treat annoying symptoms. In general, the treatment employed in myelitis may also be adopted in this disease. Great care should be exercised, however, in the administration of ergotin, since Tuczek has shown that chronic ergot poisoning may give rise to tabes. Moreover, sudden paralysis has been known to develop in these patients after the use of ergotin.

A certain amount of good may be derived from the use of electricity. The galvanic current is chiefly employed; stabile or labile spinal current,

ascending or descending; or a spinal cord-sympathetic current. If spinal pressure points are present, they may be treated with the anode. Rumpf has recently recommended the faradic brush.

If the galvanic current is employed, it should not be very strong, the sittings should not last more than ten to fifteen minutes, nor be held oftener than once a day. The electrodes should always be large in galvanization of the spinal cord.

In order to employ the spinal cord-sympathetic current, the cathode is placed immediately below and behind the angle of the lower jaw, upon the upper cervical ganglion of the sympathetic, while the anode is slowly passed, at short intervals, along the spine, at first on one side, then on the other.

In using the faradic brush, Rumpf employed a strong current ; the brush (cathode) was applied a number of times to the trunk, back, and limbs until the skin was thoroughly reddened. Duration of the sitting ten minutes, daily or every other day.

The administration of morphine and narcotics in general plays an important part in the treatment of individual symptoms.

2. *Spastic Spinal Paralysis.*

(*Lateral Sclerosis of the Cord. Tabes dorsalis spasmodica.*)

I. ETIOLOGY.—In the majority of cases this disease is a complication of various other affections of the spinal cord.

The primary form of the disease is more frequent in men than in women, and particularly from the ages of thirty to fifty years. Cold, injury, mental and bodily exhaustion, etc., are mentioned as causes. Congenital predisposition and a neuropathic taint seem to play an important part in etiology. The disease is not infrequent in childhood. The marriage of relatives and congenital syphilis seem to be causal factors.

The etiology of secondary or symptomatic spastic spinal paralysis is the same as that of the primary disease (various forms of myelitis, syphilitic spinal paralysis, spinal paralysis after acute diseases, hydromyelus, internal hydrocephalus, lead poisoning, multiple sclerosis). Spastic symptoms have even been observed in rare cases of locomotor ataxia.

II. ANATOMICAL CHANGES.—From a theoretical standpoint, Erb attributed primary spastic spinal paralysis to disease of the lateral columns of the cord. The autopsies of v. Stoffella, Morgan, Dreschfeld, and Bramwell seem to confirm this view.

v. Stoffella's case presented gray degeneration of the lateral columns, most marked in the lumbar and lower dorsal region, slightly marked in the cervical cord. The disease affected the posterior part of the lateral columns, extended internally to the posterior horns, externally to the pia mater. No microscopical examination was made.

The appearances in Bramwell's case are shown in Fig. 52 ; it is evident that the degeneration is confined to the pyramid tracts.

Westphal questions that the symptom-complex of spastic spinal paralysis is always the result of an affection of the lateral columns.

III. SYMPTOMS.—In uncomplicated cases the symptoms consist of paresis, later of paralysis of the muscles, with rigidity and contracture, exaggeration of the tendon reflexes, and spastic gait. Sensation and the functions of the bladder and rectum are unaffected.

The symptoms, as a rule, begin in the lower limbs, and gradually ex-

tend to the trunk and upper limbs. They rarely travel in the opposite direction. Still more rarely they are confined to one limb, or to the upper and lower limb of one side.

The patients first complain of a feeling of weakness in walking; this is soon followed by stiffness of the muscles during active and passive motion. Some patients complain of twitchings in the muscles. Gradually, muscular contractures develop. These affect the extensors and adductors of the thighs, the plantar flexors of the feet, the latter giving rise to pes varo-equinus. In walking, the limbs are often held as stiffly as two sticks. The patients are hardly, or not at all, able to lift the feet from the ground in walking, and therefore move the pelvis to and fro; the trunk is often bent over forward. At the same time the thighs pass each other with difficulty, on account of the contracture of the adductors. If the patient, in sitting down, brings the toes in contact with the floor, the leg will undergo trembling movements, and a

FIG. 52.

Anatomical changes in primary spastic spinal paralysis. After Bramwell. The degenerated parts in the crossed pyramid tracts are shaded. Upper dorsal region. Enlarged 10 times.

sort of hopping movement is sometimes noticed when the patient, in walking, touches the floor with the tips of the toes (vide Figs. 53 and 54).

The electrical irritability of the muscles is either unchanged or slightly diminished. The muscular contractures may become so excessive that the patient is confined to his bed. The flexor muscles may also undergo contracture, and similar changes may appear in the muscles of the trunk and upper limbs.

In uncomplicated cases, the cutaneous sensibility is unchanged. Some patients suffer from paræsthesiæ or are very sensitive to cold. There are no vaso-motor and trophic disturbances.

The cutaneous reflexes are unchanged or increased, but occasionally diminished. The tendon reflexes are always increased. This is best shown with regard to the patellar reflex. Foot clonus is also present, i. e., if the extended limb is raised, and dorsal flexion of the foot is suddenly performed by the physician, the limb undergoes rapidly succeed-

8

ng trembling motions; these may be suddenly checked, in some cases, by plantar flexion of the great toe. Reflex muscular contractions may also be produced by tapping other tendons (tendo Achillis, tendon of peronei, biceps of the arm and leg, triceps, supinators, extensors and flexors of the forearm, etc.).

The vesical, rectal, and sexual functions are unimpaired.

In the secondary forms of the disease, other symptoms, which depend upon the primary affection, may also be present.

The disease runs an acute (within a few weeks), subacute, or chronic course (more than thirty years).

IV. DIAGNOSIS.—The disease can hardly be mistaken for any other if the cardinal symptoms are taken into consideration.

V. PROGNOSIS.—In the secondary form of the disease the prognosis

FIG. 53.   FIG. 54.

Mode of walking in spastic spinal paralysis.   Mode of standing in spastic spinal paralysis.
              After Adams.   After Little.

depends upon the primary affection; in the primary form it is relatively favorable, since recovery has been observed in a number of cases.

VI. TREATMENT.—In primary spastic paralysis the best effects are obtained by the galvanic spinal current, as employed in tabes (vide page 112); in addition, warm baths (30° R.) and potassium iodide.

The treatment of the secondary form is the same as that of the disease which has given rise to it. Southam claims to have cured a case by nerve stretching.

3. Acute Spinal Atrophic Infantile Paralysis. Acute Anterior Infantile Poliomyelitis.

I. ETIOLOGY.—This disease occurs chiefly in childhood, most frequently from the age of six months to four years. It is rare in nurs-

lings before the period of dentition, though Duchenne, Jr., observed two cases in infants of twelve days and one month respectively. The disease is also rare after the age of seven years.

Boys are affected more frequently than girls. As a rule, no direct causes are demonstrable. It develops particularly during the summer; I have also seen several cases develop at the same time in adjacent localities. In fact the disease looks in many respects like an infectious disease.

Whether heredity exerts an influence in this malady is not known with certainty. Several children in a family are sometimes affected; occasionally, other children in the family have died in eclampsia. It may also occur in families in which insanity or other nervous diseases have been observed in previous generations.

The etiological influence of dentition, cold, injury, etc., has been grossly overestimated. The disease develops not infrequently during or after infectious diseases, viz., pneumonia, diphtheria, scarlatina, acute gastro-enteritis, etc.

The attack often occurs during the night without any demonstrable cause. The children go to bed in perfect health and are paralyzed during sleep. It is not true that rachitic and scrofulous children are predisposed to the disease.

II. ANATOMICAL CHANGES.—This disease is an acute inflammation of the gray matter of the spinal cord, which is localized mainly or exclusively in the anterior horns, and there gives rise to disappearance of the large motor and trophic ganglion cells.

Fig. 55.

Distribution of the foci of disease in the gray matter in acute spinal, atrophic infantile paralysis. After Roth.

a, Transverse section through the conus medullaris, 13 mm. above the filum terminale; b, transverse section through the lumbar enlargement, 10 mm. higher; c, transverse section through the middle of the lumbar enlargement, 7 mm. higher; d, transverse section through upper part of lumbar enlargement, 6 mm. higher; e, lumbar cord near the dorsal region, 7 mm. above d. Enlarged 2 times.

Leyden has recently called attention to the fact that diseases which are clinically exactly like atrophic infantile paralysis may be the result of other lesions, particularly of inflammatory processes, which are situated in the :meninges and the white matter of the cord, and extend, secondarily, to the gray matter and its large ganglion cells. In the following remarks we refer only to the typical form of the disease.

The majority of anatomical observations were made in patients who had had the disease for many years. In one case the disease had lasted only two months.

In relatively recent cases the anterior horns of the cord contain one inflammatory focus, but more frequently there are several myelitic foci, generally in the lumbar or cervical enlargements. They may be unilateral or bilateral, microscopic in size or three to four cm. in dimensions. The foci generally have a brownish-red or grayish-red color and diminished consistence.

Microscopic examination of the foci shows unusual fulness of the blood-vessels, changes in their walls, and increase of the neuroglia. The vessels present aneurismal dilatations in places; their walls contain an unusual number of nuclei and are thickened. The lymphatic sheaths contain fat granules, granulo-fatty cells, white blood-globules, rarely hæmatoidin. Granulo-fatty cells are also found, in greater or lesser numbers, in the myelitic foci. They were entirely absent in one of Leyden's cases, but were replaced by peculiar epithelioidal cells which the writer regarded as genetically identical with granulo-fatty cells. The neuroglia is increased in amount and in the number of cellular elements. The ganglion cells in the anterior horns are partly destroyed, some of them contain vacuolæ. The nerve fibres which enter the anterior horns also undergo degeneration. Charcot regards the disease as a parenchymatous myelitis, others think the connective tissue forms the starting-point (interstitial myelitis). At all events, we

Fig. 56.

Acute spinal atrophic infantile paralysis.
Transverse section through the lumbar enlargement. The middle group of cells in the right anterior horn is atrophied and sclerosed. Enlarged 10 times. After Charcot.

sometimes find foci which appear to be confined exclusively to some of the groups of ganglion cells (vide Fig. 56).

In cases which have lasted for years the myelitic foci are sclerosed. At the same time the corresponding horn diminishes in size, and the white matter often increases in consistence, and undergoes atrophy (vide Fig. 57). The ganglion cells of the anterior horns have often disappeared in places, or those which remain contain yellow pigment, are atrophied, or may present amyloid degeneration or sclerotic distention (Rosenthal).

The poliomyelitic foci are sometimes converted into cystic spaces, which are traversed by a network of connective tissue.

Degenerative changes develop not infrequently in the lateral columns,

occasionally even in the anterior columns. Clark's columns were affected in some cases.

The posterior nerve roots are unchanged; the anterior roots corresponding to the diseased portion of the cord undergo atrophic changes. They appear thin and gray, and the microscope shows disappearance of nerve fibres and increase of the interstitial tissue. Similar changes are also found in the peripheral nerves.

The paralyzed muscles often have a strikingly pale-red color; at a later period this sometimes changes to a brownish-red. In the early stages the microscope shows diminution in the size of the fibres and increase of the nuclei of the sarcolemma (Leyden found a few hypertrophic fibres among the atrophic ones); in some fibres there are fissures resembling those of Zenker's typhoid degeneration of the muscles. At a later period the transverse striation becomes obscured, and fat granules make their appearance. The interstitial connective tissue increases in amount

Fig. 57.

Acute spinal atrophic infantile paralysis.
Transverse section through the cervical cord. Right anterior horn atrophic and sclerosed, the white columns diminished in size on same side. Paralysis of right arm during life. Death 50 years after the attack of paralysis. Enlarged 10 times. After Charcot.

and in the number of nuclei. The adipose tissue sometimes increases to such an extent that the volume of the muscle seems to have enlarged. Upon section, such muscles look like a mass of fat, in which are seen a few pale streaks of relatively normal muscular tissue. In other cases the muscles are converted into thin, connective-tissue, tendon-like strands. In one case Déjérine found complete atrophy of the intermuscular nerves and nuclear proliferation in the sheath of Schwann.

The panniculus adiposus of the paralyzed limb, as a rule, is unusually well developed; but the vessels, ligaments, fasciæ, and bones undergo atrophy. The thickness of the bones, particularly of the compact substance, is diminished, and they are sometimes as flexible as in osteomalacia.

In one case, Sander described slight development of the central convolutions of the brain, but the disease was associated with idiocy.

III. Symptoms.—The symptoms consist of the sudden occurrence of completely developed, non-progressive, flaccid paralysis, with rapid atrophy of the paralyzed muscles, rapid development of degeneration reaction in the paralyzed muscles and nerves, abolition of the cutaneous and tendon reflexes, intact sensation, bladder, and rectum, and absence of trophic changes in the skin.

The paralysis sometimes occurs unnoticed, sometimes it develops with febrile and cerebral symptoms.

In some cases the paralysis is discovered accidentally. The patients have perhaps had some illness, and when they attempt to leave the bed, they are found to be unable to walk. Or the child is brought to the physician because it learns to walk very late, uses the hands awkwardly, etc., and the most superficial examination then shows severe paralyses and deformities. In many cases it is said that the children have gone to bed well, and were found to be paralyzed on waking the next morning. In other cases, epileptiform convulsions occur, either unexpectedly or after several days of general malaise, anorexia, restlessness, etc. The patients lose consciousness, become convulsed, are sometimes bathed in perspiration, and have a high fever. The spasms may last one or more hours, in rare cases recur at definite intervals, and are often looked upon as the result of teething. When the convulsions disappear, the weakness or paralysis of the muscles at once becomes noticeable. The fever may last several days after the spasms have ceased. It generally lasts two to three days, rarely more than a week; it ranges between 39–40° C.

The paralysis affects the lower limbs much oftener than the upper ones; there is often paraplegia of the lower limbs. Occasionally the arm and leg on the same side are paralyzed, or the arm on one side, the leg on the other; in rare cases both upper limbs are paralyzed. The muscles of the trunk, back, scapulæ, more rarely the neck, may also be involved. In very exceptional cases the facial muscles are attacked.

In many cases the disease at first attacks all the muscles of a limb, but in the next few days or weeks the paralysis disappears in most of the muscles, but persists in others. In very rare instances the paralysis disappears entirely (temporary paralysis).

Careful observation shows that the muscles are very often affected in certain definite groups. This indicates that certain parts of the cord are apt to be affected, and that, on the other hand, spinal centres for definite groups of muscles are situated in close proximity in such localities. E. Remak distinguishes two types of the disease in the upper limbs : a, the arm type, involving the deltoid, biceps, brachialis internus, and the supinators; b, the forearm type, affecting chiefly the extensors, and leaving the supinators intact. The centres for the extensors and flexors of the forearm probably are relatively remote from one another, the former in the middle of the cervical enlargement (fourth and fifth cervical nerve roots), the latter lower (eighth cervical and first dorsal nerves). In paralysis of the distribution of the crural nerve, the sartorius generally escapes, while in paralysis of the quadriceps femoris the tibialis anticus is not infrequently also affected ; the latter muscle generally escapes in spinal paralysis of the peroneal nerve. These phenomena also indicate a peculiar arrangement of the muscle centres in the cord. According to Kahler and Pick, the spinal centre of the calf muscles is situated near the fourth and fifth dorsal roots.

The electrical reactions of the paralyzed muscles and nerves present very important changes, viz., those of degeneration reaction (vide page 8).

The electrical changes appear within a few days after the development of the disease.

The paralyzed muscles are often tender on pressure. Cutaneous hyperæsthesia is sometimes observed at the onset of the disease.

The paralyzed muscles rapidly undergo very marked atrophy. This can sometimes be followed distinctly from week to week. The disappearance of the muscles is sometimes marked by an abundant deposit of fat. Fibrillary twitchings in the atrophic muscles have often been described.

The panniculus adiposus generally becomes unusually abundant, so that it may be difficult to feel the thin bellies of the muscles. The skin is generally very cool, bluish-red and marbled in appearance, sometimes œdematous and fissured, or covered with cold perspiration. A tendency to ulceration and the development of frost-bites is mentioned by some writers.

The difference of temperature between the healthy and paralyzed limbs may exceed 15° C. According to Tartière, the paralyzed limb, during the initial febrile period, is 1–2° C. warmer than the corresponding healthy limb; later the limb gradually grows cooler.

If the paralysis is extensive, the cutaneous and tendon reflexes are entirely abolished within the domain of the paralysis.

The bladder and rectum are unaffected; at the most, there is incontinence at the beginning of the disease, rarely retention of urine and constipation.

The atrophic changes extend to the bones, fasciæ, tendons, etc. The pulse of the paralyzed limbs has been known to become smaller. If the disease has begun in early childhood, the paralyzed parts often remain as mere rudiments. The difference in length of the corresponding bones on the healthy and paralyzed sides may exceed twenty centimetres, but, as a rule, it is much less. Seeligmueller has observed, in a few cases, unusual length of the bones in the paralyzed limbs.

The disease terminates with the development of deformities. Several factors concur in their production. In some cases they are the result of contracture of the non-paralyzed muscles. In other cases, however, the paralyzed muscles themselves undergo contracture, and the action of gravity upon the limbs then aids in producing deformity.

In the foot we generally find pes equinus or varo-equinus, rarely pes valgo-equinus or calcaneus. At the knee we not infrequently find genu recurvatum, i. e., the leg can be extended to a much larger angle than normally. Contracture at the hip-joint is a rarer sequel. Scoliosis, lordosis, rarely kyphosis, may result from partial paralysis of the muscles of the back. Contracture of the pectoral and latissimus dorsi sometimes impairs the mobility of the shoulder-joint. Or, on account of paralysis of the deltoid, the shoulder-joint becomes loose, inasmuch as the weight of the arm draws it downwards; the capsule of the joint is relaxed and a deep furrow is often visible between the spine of the scapula and the head of the humerus. Contracture at the elbow-joint is rare; it is more frequent in the hand and fingers.

The onset of atrophic infantile paralysis is almost always acute; in rare cases it runs a subacute course, and the paralyses do not develop for several days. Acute exacerbations followed by fresh paralyses are sometimes observed. Life is rarely, if ever, endangered.

IV. DIAGNOSIS.—The differential diagnosis must take into consideration :

a. Progressive muscular atrophy; this hardly ever occurs in childhood, except in the hereditary form; it develops very slowly.

b. Pseudo-hypertrophy of the muscles; this develops very gradually, and muscular atrophy remains absent.

c. Spastic spinal paralysis; muscular atrophy and degeneration are not produced, and the tendon reflexes are increased.

d. Parturition paralyses; these are present from birth, labor has usually been difficult; sensory disturbances are generally noticeable.

e. In acute central or transverse myelitis, in hæmatomyelia and compression myelitis, sensory disturbances are generally present; there are often disturbances of the bladder and rectum, trophic changes in the skin, increased cutaneous reflexes, absence of degeneration reaction.

f. Multiple degenerative neuritis is generally associated with sensory disturbances.

V. PROGNOSIS.—The prognosis is not especially favorable. As a rule, there is no danger to life, but we are unable to relieve the paralysis.

VI. TREATMENT.—During the period of the initial fever and spasms, we may order a warm bath (28° R.), lasting half an hour, with cold douches, and then an ice-bag to the head.

After the paralysis makes its appearance, the Chapman ice-bag may be applied to the supposed site of disease in the cord. Leeches, cups, and derivatives of all kinds have also been applied. Potassium iodide may be given internally to aid absorption of the inflammatory foci; ergotin and belladonna have also been given for the same purpose.

After the most acute symptoms have subsided, we may attempt to aid absorption by the use of the galvanic current (large electrodes), at first the anode (two to three minutes), then the cathode over the site of disease.

The paralyzed parts are treated with the galvanic or faradic current, the latter only when the muscles respond. The electrodes should be thoroughly moistened, and strong currents should be used in order to penetrate the skin and panniculus adiposus. In a number of cases we employed, at the same time, the galvanic spinal current and peripheral faradic stimulation of the muscles.

Gymnastics, massage, tenotomy, and orthopædic instruments may be employed to prevent and relieve deformities.

4. *Acute, Subacute, and Chronic Spinal Atrophic Paralysis of Adults.*

Acute, subacute, and chronic anterior poliomyelitis of adults.

These morbid processes are in part identical with, in part very closely allied to, acute atrophic infantile paralysis.

a. Acute spinal atrophic paralysis of adults corresponds entirely to the similar disease in childhood. Men are attacked more frequently than women; it generally occurs before the age of thirty years. Among the causes adduced are cold, overexertion, and excesses in Baccho et Venere. It has been observed a number of times after infectious diseases in women, particularly after puerperal fever. A certain etiological influence is sometimes attributed to heredity, tuberculosis, and syphilis.

The first symptoms are those of a general febrile affection, but convulsions are not produced. It may begin with a violent chill, followed by high temperature, headache, delirium, sometimes vomiting and diarrhœa, often followed by a typhoid condition. Bramwell described temporary aphasia in one case. The fever generally lasts a week, some-

times more than two weeks. The patients often complain of pain in the back and limbs.

More or less extensive flaccid paralysis makes its appearance in a few hours, occasionally in a single night, more rarely in the course of several days. This may be confined to a single group of muscles, or an entire limb; it sometimes appears as hemiplegia, paraplegia, or crossed paralysis; it rarely attacks the muscles of the neck, hardly ever those of the face. In a few cases the respiratory muscles were affected, and the disease then ended fatally by producing suffocation.

In a few cases the paralysis gradually disappears in a few weeks or months (temporary paralysis); in others the paralysis disappears in some muscles, but persists in others. The paralyzed muscles are often sensitive on pressure, and rapidly undergo atrophy; degeneration reaction is also produced. As a matter of course, the growth of the bones is not interfered with, as in children, but contractures of the muscle and, to a less extent, deformities are produced. The skin of the paralyzed parts is cold, often bluish-red, and does not perspire; it is sometimes œdematous. Sensibility is retained, but paræsthesiæ and cutaneous hyperæsthesia are sometimes present at the outset. Reflex irritability is abolished in total paralysis of a limb, otherwise it is diminished or unchanged. The functions of the bladder and rectum remain normal, though vesical paralysis is occasionally noticed at the outset. The sexual powers are unaffected.

The prognosis as regards life is generally favorable, although a fatal termination is possible. The remarks made concerning the anatomical changes, diagnosis, and treatment of this disease in children also hold good in adults.

b. Subacute and chronic spinal atrophic paralysis of adults occurs at a more advanced age (thirty to fifty years), but Erb recently observed a case in a child of six years. The causes are sometimes the same as those of the acute form, sometimes no cause can be discovered. Remak showed that lead poisoning may give rise to this disease.

It generally begins gradually without any severe impairment of the general condition. The patients tire easily in walking, and, finally, well-marked paralysis develops. As a rule, it begins in the lower limbs, and then extends along the trunk to the upper limbs; more rarely the paralysis progresses in the opposite direction. It may also be confined to a few groups of muscles. It begins most frequently in the extensors of the legs; in the upper limbs, likewise, the extensors of the forearms are first affected. The paralysis gradually extends from one group of muscles to another. According as this occurs in the course of weeks or of months and years, the disease is called subacute or chronic. The muscles of the neck are not infrequently affected. The disease may even extend to the bulbar nuclei, bulbar symptoms then develop, and death occurs from suffocation, etc. The character of the paralysis (apart from its progressive quality) is the same as that of acute atrophic paralysis, *i. e.*, the paralysis is always flaccid. It may entirely disappear in the course of weeks or months, or it disappears only in part while other muscles undergo rapid atrophy. This may be followed by contracture and a hard tendinous condition of the muscles. The electrical irritability is the same as in infantile paralysis; in the beginning, the mechanical irritability of the muscles may be increased.

Improvement generally follows an opposite course to the paralysis, *i. e.*, first in the arm, then in the legs; the muscles supplied by the pero-

neal nerve often remain paralyzed. Fibrillary contractions are often observed in the paralyzed muscles.

Some patients complain of paræsthesiæ, but the cutaneous sensibility is objectively unchanged. Disturbances of micturition are sometimes observed at the beginning of the disease, but they disappear at a later period. The joints and sheaths of the tendons are sometimes swollen. The cutaneous and tendon reflexes are abolished if a limb is completely paralyzed, otherwise they are diminished or unchanged. In progressive cases death occurs in one to four years.

Authors are agreed that the lesion consists chiefly of destruction of the ganglion cells of the anterior horns; atrophy of the cells of Clark's columns, and of the anterior portions of the posterior horns, is also mentioned.

The neuroglia may be unchanged or increased; there may also be proliferation of the nuclei in the walls of the vessels, and atrophy of the fibres passing out of the anterior horns and of the anterior nerve roots. Degenerative changes have also been observed in the columns of the cord, the peripheral nerves, and paralyzed muscles.

The diagnosis is not always easy, especially in old cases. It is distinguished from progressive muscular atrophy by the following features: the paralysis precedes the atrophy, it is not fascicular, but affects the muscles *en masse ;* in progressive muscular atrophy, the reflexes are retained, the disease is slower, but less unfavorable as regards the maintenance of life, recovery does not occur, and degeneration reaction occurs only in those parts of the muscles which are affected with special intensity.

In amyotrophic lateral sclerosis, muscular rigidity and spasms, and increased tendon reflexes are noticeable. Multiple degenerative neuritis is attended with sensory disturbances.

The prognosis is favorable as regards life, but less favorable as regards complete recovery.

The treatment is the same as that of the acute form.

5. *Progressive Spinal Muscular Atrophy.*

(*Amyotrophia spinalis progressiva. Polyomyelitis anterior chronica disseminata.*)

I. Etiology.—In spinal progressive muscular atrophy, there is a progressive disappearance of voluntary muscles, beginning in the upper limbs, and first affecting only parts of the muscles (fascicular), and unattended with paralysis, apart from the loss of power due to the disappearance of the muscular fibres. There are no disturbances of sensation, or of the functions of the bladder and rectum. All these changes are the results of a primary chronic affection of the anterior columns of the cord, particularly of the large ganglion cells.

As a rule, the disease occurs between the ages of 30 and 50 years. Cases have also been reported in which the disease began in childhood—especially when it is propagated in families from one generation to another—but the belief is now growing that these cases are really due to a primary affection of the muscles. We shall, therefore, discuss them under the head of juvenile muscular atrophy, or dystrophia musculorum progressiva.

Heredity is often mentioned as a cause of progressive muscular atrophy, but this applies more particularly to the juvenile form.

The disease is often connected with overstrain of certain muscles and groups of muscles.

In other cases it is attributed to injury, cold, onanism, and sexual excesses.

It has been claimed that the malady occurs after infectious diseases (measles, scarlatina, small-pox, cholera, acute articular rheumatism, typhoid and puerperal fever, syphilis; according to Jackson and Clark, even after vaccination), but it has probably been mistaken for paralysis and atrophy resulting from myelitis, neuritis, or changes in the muscles themselves. This is also true of the injurious effects of lead and arsenic.

Progressive muscular atrophy is more frequent in men than in women, probably because the former are more exposed to the etiological influences.

II. SYMPTOMS.—Atrophy and, in connection with it, weakness of the muscles develop very gradually. As a rule, this begins in the interossei and the muscles of the thenar and hypothenar eminences, so that

FIG. 58.　　　　　　　　　　FIG. 59.

Right hand.　　　　　　　　Left hand.

Dorsum of hand with depressed interosseous spaces in progressive muscular atrophy, in a woman æt. 34 years.

the patients first complain of stiffness in the fingers and inability to perform delicate manipulations. These disturbances are increased by cold and diminished by warmth.

According to Eulenburg, the first interosseus is one of the very first muscles affected. Atrophy of the interossei is shown by the deep grooves in the interosseous spaces (vide Figs. 58 and 59). The ball of the little finger and thumb becomes flattened, and the subjacent bones can be felt more distinctly (vide Figs. 60 and 61). In addition, the fingers assume unusual positions. The muscles first attacked in the ball of the thumb are the adductor and opponens pollicis. On account of the predominance of the extensors and abductors of the thumb, the latter is brought into a position of abduction and dorsal flexion (ape-hand). Atrophy of the interossei gives rise to the development of the so-called claw-hand, as the result of the predominance of the lumbricales; the first phalanges are hyper-extended, the second and third phalanges are strongly flexed. The claw-hand also develops in other affections, for

example, in traumatic ulnar paralysis, but in this disease the middle and ring fingers are most markedly affected (vide Figs. 62 and 63).

If the lumbricales undergo atrophy the palm of the hand is flattened, but the extensor digitorum continues to extend the first phalanges. The

FIG. 60.   FIG. 61.

Right hand.   Left hand.

Palm of hand with atrophy of thenar and hypothenar eminences. The same patient.

disease sometimes jumps from the muscles of the hand to the deltoid. In this muscle it is especially evident that the atrophy occurs in bundles, not *en masse*. At first the posterior and middle thirds are chiefly

FIG. 62.   FIG 63.

FIG. 62.—Claw hand in progressive muscular atophy. After Duchenne.
FIG. 63.—Claw hand in paralysis of the interossei as the result of injury to the ulnar nerve.

affected. When the entire muscle has undergone atrophy, the shoulder looks flat and angular, and the edges of the bones appear very distinctly. As a matter of course, the movements of the arm are interfered with, and,

if the scapular muscles are also attacked, the arm hangs alongside the trunk almost like an inert mass.

If the ligaments of the shoulder-joint are very flaccid, subluxation of the head of the humerus may result.

In other cases the disease spreads from the finger muscles to those of the forearm, especially the extensors. The supinators usually escape unless the flexors become involved. The atrophy may be so extensive that the forearm consists of hardly anything beyond skin and bones, and the interosseous space forms a deep groove (vide Figs. 64 and 65).

Among the muscles of the arm the triceps is the last to be affected, so that the arm is almost always extended.

FIG. 64.   FIG. 65.

Appearance of the hand and forearm in two cases of progressive muscular atrophy. Dorsal surface. After Duchenne.

The right hand is generally the first attacked, more rarely the left hand or both at the same time. In the majority of cases, after the muscles of one side of the body have been attacked, the corresponding ones on the other side are involved before the morbid process extends to higher or lower groups of muscles.

If the disease is regarded as spinal in its origin, it must be assumed that the process extends from one-half of the cord to the other before it spreads in a longitudinal direction.

As a rule, the muscles of the scapula and trunk are not attacked until the arms have undergone considerable emaciation. According to Du-

chenne, they are attacked in the following order: lower part of the trapezius, pectorals, latissimus dorsi, rhomboid, scapularis, extensors and flexors of the head, deep muscles of the back, abdominal muscles, finally the neck muscles and rotators of the head. Here, also, the disease often begins on one side, or is more marked on one side than on the other. If the muscles of the loins are affected, the spine is unusually curved anteriorly, so that, in the vertical position, a vertical line dropped from the shoulder falls behind the sacrum (vide Fig. 66). If the abdominal muscles are atrophied, a slight bronchitis may prove dangerous, inasmuch as the energy of coughing is interfered with, the bron-

FIG. 66.   FIG. 67.

Position of the body in atrophy of the lumbar muscles in progressive muscular atrophy. After Duchenne.

Position of the body in paresis of the abdominal muscles in progressive muscular atrophy. After Duchenne.

chial secretion may accumulate, and death occur from suffocation. Feebleness of the abdominal muscles also causes marked projection of the spine anteriorly, but a vertical line dropped from the shoulder passes through the sacrum (vide Fig. 67). The diaphragm sometimes takes part in the atrophic process.

The lower limbs are usually attacked last. The flexors of the legs are affected earliest and most markedly.

Fibrillary (more properly fasicular) muscular twitchings are an almost constant symptom of progressive muscular atrophy. They consist of repeated brief contractions which always affect only individual

parts of a muscle, now here, now there. They are especially active on disrobing, blowing on, tapping or faradizing the muscles. They sometimes appear in muscles which do not present any visible emaciation, but may be entirely concealed by a fat integument. In some cases they are so vigorous as to cause involuntary twitchings of the fingers, arm, or leg.

The electrical irritability of the paralyzed muscles corresponds, in general, to the degree of atrophy. The more the muscular substance is lost, the more the electrical irritability of that muscle diminishes until it is entirely abolished. Careful examination, however, shows that partial, then complete degeneration reaction is manifested in parts of the muscle.

If a small anode is placed in the mastoid fossa or adjacent part of the neck, and a large electrode between the scapulæ, more or less vigorous movements will be produced in the arm on the side opposite to the anode. This phenomenon, to which the term diplegic contractions has been applied, is observed at times in this disease, but is by no means characteristic.

If the muscles of a limb are extensively affected, the integument is often bluish-red, marbled, and feels cold; its temperature has been found markedly diminished (5° C.). Some writers have observed an increase of temperature (0.5° C.) at the beginning of the disease. The skin presents a tendency to trophic changes: thickening, formation of scales, diminished growth of hair, tendency to ulcerations, herpetic and pemphigoid eruptions, thickening of the nails, etc.

An abnormally abundant production of sweat has also been observed, particularly in rapidly progressing or far advanced cases. Swelling of the joints and phalanges is sometimes described.

Sensory cutaneous disturbances are almost always absent; at the most the patients complain of a feeling of coldness in the limbs. The cutaneous and tendon reflexes disappear more and more with the extension of the disease.

The functions of the bladder and rectum are unaffected.

Oculo-papillary changes were often noticed. The pupils were unusually narrow on one or both sides, reacted slowly to light, and lost the ability to dilate.

Landouzy mentions changes in the ocular muscles in one case. Impaired sight and flattening of the cornea were also mentioned in one case.

The general condition is very little affected; slight febrile movement occurs occasionally, and is explained by Friedreich as absorption fever, the result of the absorption of chemical products of the muscular degeneration.

The disease advances very slowly and, as a rule, lasts many years. The patient finally loses the use of his hands and arms, is unable to move his legs, and becomes utterly helpless. The mental powers generally remain intact.

The condition becomes even more distressing when it is complicated with bulbar symptoms; atrophy and paralysis of the muscles of the face, tongue, pharynx, and larynx. In some cases the latter symptoms first develop, and are followed by the symptoms of progressive muscular atrophy.

Death occurs from intercurrent diseases or increasing marasmus, or

bulbar symptoms give rise to difficulty in deglutition, inanition, "foreign-body" pneumonia, etc.

III. ANATOMICAL CHANGES.—According to Charcot, progressive muscular atrophy is dependent on a primary chronic destruction of the large ganglion cells in the anterior horns of the spinal cord. The French writers attribute these changes to inflammatory lesions of the ganglion cells, which give rise to pigment degeneration and atrophy, or to sclerotic atrophy and complete disappearance of the cells. Any changes which may be present in the interstitial tissue (nuclear proliferation, dilatation, thickening, and fatty degeneration of the blood-vessels) are said to be merely secondary. Others maintain that the process begins in the interstitial tissue, or that both modes of origin are possible. If the lesion is well marked, the corresponding anterior horn, and even the entire half of the cord are diminished in size (vide Fig. 68). Degeneration has also been noticed in the white columns, particularly the lateral columns.

FIG. 68.
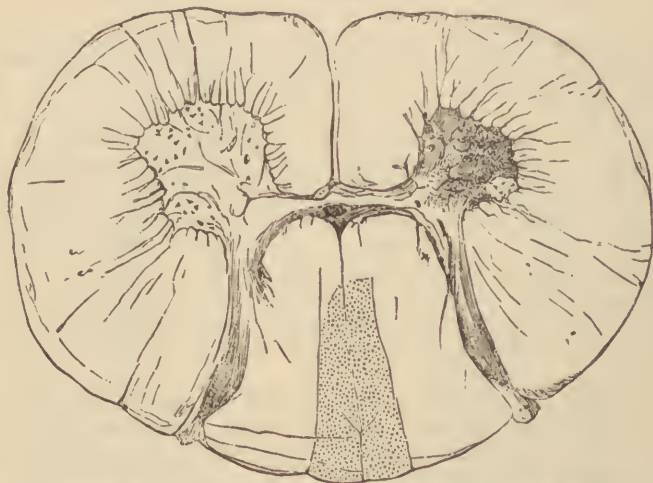
Transverse section of the cervical cord in progressive muscular atrophy.

The right anterior horn and right half of the cord are diminished in size, the ganglion cells of the right anterior horn have disappeared with the exception of a few cells in the external posterior angle. Enlarged 10 times. After Charcot.

The anterior nerve roots are often thin, gray, and in a condition of fatty degeneration and atrophy. Degenerative changes have also been observed in the peripheral nerves.

Friedreich's examinations concerning the muscular changes have furnished the following results: They begin in the intermuscular connective tissue (perimysium internum), with increase in amount and nuclear proliferation; this is followed directly by changes in the muscular substance; the nuclei of the sarcolemma proliferate; there is gradual disappearance of the muscular substance proper; some fibres undergo waxy degeneration, others cloudy swelling; some fibres are split longitudinally, others in transverse sections; finally, some of the fibres appear split up into their elementary constituents. At the same time the muscle substance gradually disappears, so that, in numerous places, the sarcolemma contains nothing but nuclei. These changes often occur only

in certain parts of the muscle. The atrophic muscle appears pale, occasionally brownish-red (unusual amount of pigment in the fibres). Finally, it may be converted into a sort of tendinous structure (fibrous degeneration). Fatty degeneration of the muscular fibres and accumulation of fat in the interstitial connective tissue are rare, but are sometimes so extensive as to cause increased size of the muscle.

IV. DIAGNOSIS.—The diagnosis is usually easy if we bear in mind the beginning of the disease in the ball of the thumb and little finger, and in the interossei, the absence of sensory disturbances, the development of fascicular atrophy, and then of weakness of the muscles, the fibrillary contractions, and the intact condition of the bladder and rectum.

The disease may be mistaken for:

a. Juvenile progressive muscular atrophy ; this is often hereditary and generally begins in childhood; it often begins in the lower limbs. Duchenne sometimes noticed a pecular impairment of motion of the lower lip in the beginning of the disease. Moreover, the juvenile atrophy is much slower than the spinal form, fibrillary contractions are usually absent, the atrophic muscles often increase in size from the interstitial proliferation of fat, and the affected muscles do not present the degeneration reaction.

b. Multiple degenerative neuritis, when very extensive and slow in its course, may also resemble progressive muscular atrophy. The differential points are: the occurrence of paralysis prior to the atrophy, the occurrence of pain, paræsthesiæ and objective sensory disturbances of the integument; the affected muscles and nerves are often tender on pressure.

c. Acute, subacute, and chronic poliomyelitis are also characterized by paralysis, muscular atrophy, degeneration reaction, absence of sensory disturbances ; but the process does not extend in such a typical manner as in spinal progressive muscular atrophy, and, in addition, the paralysis develops first, the atrophy later.

d. Muscular atrophy is sometimes secondary to other diseases of the spinal cord, and occurs whenever myelitis, tabes, multiple sclerosis, tumors, etc., affect the ganglion cells in the anterior columns of the cord. The differential diagnosis depends upon the fact that the muscular atrophy is preceded by other signs of spinal disease.

V. PROGNOSIS.—The prognosis is not favorable, since we are hardly able to restore the atrophied muscles to the normal condition and to prevent the spread of the disease. The more extensive and rapid the atrophy the more gloomy the prognosis as regards danger to life. Bulbar symptoms generally accelerate the fatal termination. Under other circumstances life may be prolonged for many years.

VI. TREATMENT.—Overstrain of muscles must be avoided and, if the signs of muscular atrophy make their appearance, the injurious occupation must be abandoned forthwith.

The remedies recommended in myelitis (vide page 88) have been employed in this disease, but very little can be expected from them. The cautious employment of massage and rational gymnastics may be recommended. Electricity remains our chief remedy. The most suitable mode of application is the galvanic spinal current (large electrodes, the anode for the first three to five minutes, then the cathode over the site of disease, or the ascending and descending spinal current). This may be followed by peripheral galvanization of the indi-

9

vidual muscles, the cathode labile to the muscles, the anode on an indifferent spot, or over the lumbar or cervical enlargement, according as the application is made to the lower or upper limb. Galvanization of the cervical sympathetic has also been recommended. The muscles may be treated peripherally with a not too strong faradic current. Treatment requires patience on the part of the physician as well as the patient.

COMBINED SYSTEM DISEASES OF THE SPINAL CORD.

1. *Hereditary Ataxia.*
(*Friedreich's Disease.*)

This disease is pre-eminently hereditary. For example, Friedreich observed nine cases in three families; Immermann and Ruetimeyer described eleven cases in two families. Unlike ordinary tabes, the female sex is more often affected.

The first symptoms begin at the age of 4 to 7 years, or at the period of puberty. Ataxia is a prominent symptom from the beginning, while sensory disturbances remain absent or do not develop until very late; lancinating pains are rare. The ataxia extends very rapidly to the upper limbs, or it affects the upper and lower limbs at the same time. Ataxia of the ocular muscles (ataxic nystagmus) and disturbances of articulation (ataxia of the muscles of speech) may also occur. The Brach-Romberg symptom was observed in a number of cases. The ocular symptoms (ocular paralyses, reflex rigidity of the pupils, amaurosis), so common in ordinary ataxia, are wanting. The vaso-motor and secretory symptoms observed are polyuria, salivation, and hyperidrosis. No disturbance of the bladder, no bedsores; the patellar reflex absent. At a later period, the muscles become paralyzed and contractured.

In a fatal case, degeneration was found, not alone in the posterior columns (most markedly in the cervical cord), but also in the lateral columns and, in the cervical portion, in the anterior columns (vide Fig. 69).

The treatment is the same as that of tabes dorsalis.

FIG. 69.

Anatomical changes in the cord in Friedreich's hereditary ataxia. The diseased parts are shaded. *a–c*, cervical cord; *d*, dorsal cord; *e–g*, lumbar cord. After Friedreich.

2. *Secondary Degeneration of the Columns of the Spinal Cord.*
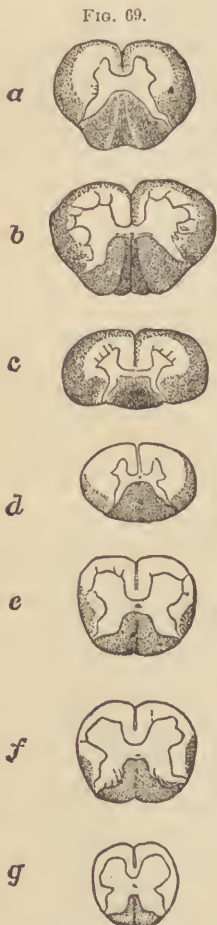
We distinguish ascending, descending, and combined secondary degeneration in the spinal cord.

a. Descending secondary degeneration of the columns of the spinal cord follows diseases of the brain, and occupies the domain of the pyramid tracts. Hence the changes are found in the posterior part of the lateral columns (the so-called lateral column-pyramid tract), and in the inner-most parts of the anterior columns, next to the anterior longitudi-nal fissure (vide Fig. 70, 1 *pvs* and 3 *psb*). Both fields contain nerve tracts which convey the stimulus of the will from the brain to the peri-phery.

As the cerebral lesion generally affects only one side of the brain, the degeneration in the cord is also found only in one pyramid tract. But since the lateral column-pyramid tract contains fibres which decussate in

Fig. 70.
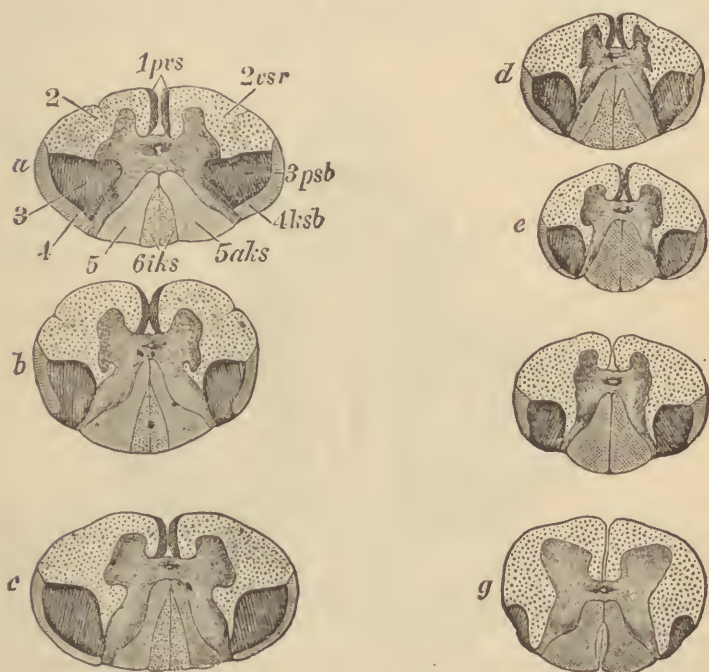
Schematic representation of the distribution of the principal tracts in the spinal cord. 1 *pvs*, anterior column-pyramid tract (undecussated) ; 2 *vsr*, antero-lateral column (divided into anterior fundamental column and lateral column) ; 3 *psb*, lateral column-pyramid tract (de-cussated) ; 4 *ksb*, lateral cerebellar tract ; 5 *aks*, columns of Burdach ; 6 *iks*, columns of Goll. Enlarged 2 times. *a*, Level of first cervical nerve ; *b*, third cervical nerve ; *c*, sixth cervical nerve ; *d*, third dorsal nerve ; *e*, sixth dorsal nerve ; *f*, twelfth dorsal nerve ; *g*, fourth lumbar nerve.

the pyramids, while the pyramid-anterior column tract does not decussate, the secondary degeneration in the former will be found on the side op-posite to the cerebral lesion, in the latter on the same side.

It must not be forgotten that, according to Flechsig's researches, there are remarkably great individual differences in the proportion be-tween the decussated and non-decussated fibres of the pyramid tracts. The decussation is sometimes complete, so that no degeneration can occur in the anterior columns. On the other hand, the degeneration in the anterior column may be unusually marked, because very slight decussa-

tion has taken place in the pyramids. Pitres reports four cases in which degeneration of both lateral column-pyramid tracts followed a unilateral lesion of the brain.

As a matter of course, not all diseases of the brain are followed by secondary degeneration of the cord. This only occurs when the pyramid tracts have been interrupted in any part of their course. If these tracts are followed centrally from their decussation, they will be found to pass through the pons into the middle and inferior parts of the pedunculus cerebri, then to the posterior division of the internal capsule, and thence to the motor regions of the cerebral cortex (anterior and posterior central convolutions). Diseases of other parts of the cortex or brain will not be followed by secondary degeneration of the spinal cord.

If the pyramid tract is affected in the cerebrum, the secondary degeneration is noticed in the peduncle, pons, and the corresponding side of the decussation in the medulla (vide Fig. 71). The middle (more

FIG. 71.
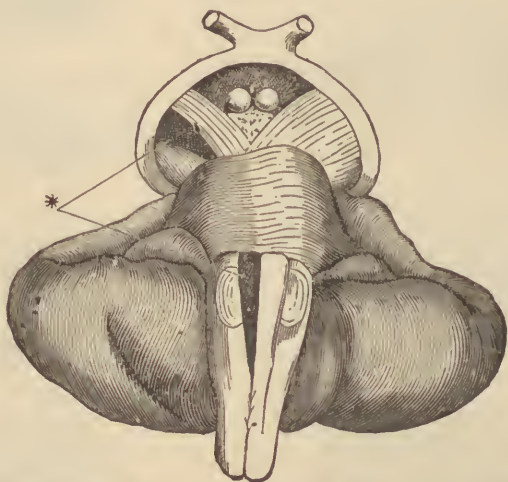
Secondary degeneration of the cerebral peduncle, pons, and medulla oblongata. The shaded parts marked * are degenerated. After Charcot.

rarely also the internal) portion of the peduncle appears gray, transparent, grayish-yellow or red, and occasionally atrophic (vide Fig. 72, *a*). The pons is also atrophied; but here the degenerated pyramid tract is visible only on transverse section, upon which it appears gray and reduced in size when compared with the intact side (Fig 72, *b*). The pyramid is also gray and diminished in size, so that the olivary body is more distinct than its mate. The changes in the spinal cord can be only followed in transverse sections, and in many cases microscopical examination is necessary. If the cord is hardened in chromic acid or bichromates, the degenerated portions assume a light-yellow color, but the yellow region is often larger than the degeneration itself. The degenerated parts stain very deeply with carmine and aniline colors, on account of the abundance of connective tissue and the dearth of nerve-fibres.

The shape and extent of the foci are shown in Fig. 72, *e* to *h*. The degeneration of the pyramid-anterior column tract diminishes more and more in its downward course and ceases in the upper part of the lumbar

cord. The degeneration in the lateral column (on the opposite side) is most extensive in the cervical cord, where it has a triangular or wedge-shape; in the dorsal cord it is less extensive and round. In the lumbar cord it again becomes triangular, and gradually approaches the surface of the cord until it is immediately beneath the pia mater. It terminates at the level of the third and fourth sacral nerves (Fig. 70).

FIG. 72.
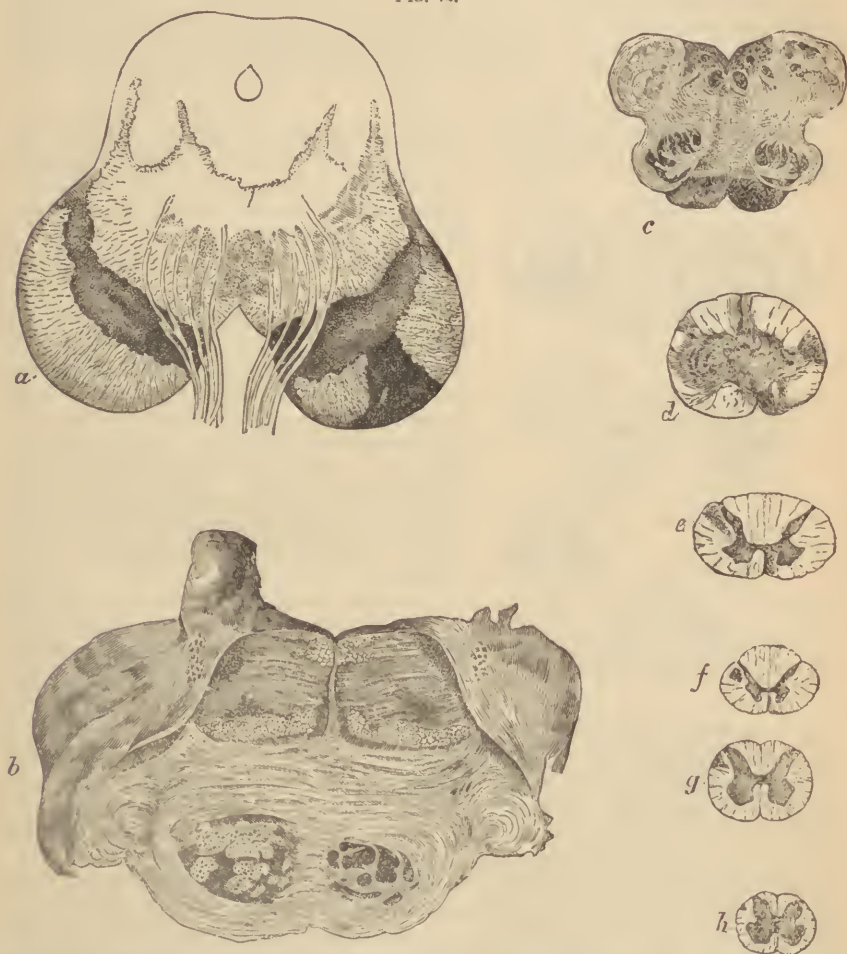
Transverse sections of the cerebral peduncle, pons, medulla oblongata and spinal cord, in a condition of secondary degeneration (shaded portions) following a lesion in the right cerebral hemisphere; *a*, peduncle; *b*, pons; *c*, medulla oblongata; *d*, decussation of the pyramids; *e*, cervical enlargement; *f*, dorsal cord; *g*, lumbar cord; *h*, conus medullaris.

The first changes in descending degeneration appear in the nerve fibres themselves. Kahler and Pick detected them on the eleventh day after an injury to the brain, and perhaps they begin even earlier. Its rapid extension, and its restriction to definite systems of fibres, precludes the idea of a propagated inflammation, and justifies the conclusion that we have to deal with a degenerative atrophy, the result of a separation of the nerve fibres from their trophic centres.

The degenerated fibres undergo loss of the medullary sheath and, finally, of the

axis cylinders; according to Henle, atrophy of the axis cylinders precedes disappearance of the medullary sheaths. At a later period, the interstitial connective tissue increases secondarily in amount, granulo-fatty cells and amyloid bodies appear in it, the walls of the vessels become thickened, and their lymphatic sheaths contain fat granules and granulo-fatty cells.

As a rule, the gray matter is unchanged. In rare cases, the anterior horn on the side opposite to the cerebral lesion is diminished in size, and its ganglion cells are atrophied, corresponding to atrophy of the paralyzed muscles during life.

If the changes have lasted a long time, proliferation of the interstitial connective tissue, and disappearance of the nerve fibres may take place in the peripheral nerves of the limbs which have been paralyzed as the result of the cerebral lesion, and the corresponding muscles may present diminished size, fatty degeneration or proliferation of the nuclei of the sarcolemma, and increase of the interstitial connective tissue, or proliferation of fat.

The contractures to which hemiplegics become subject, and the exaggeration of the tendon reflexes, are attributed to the secondary degeneration. The prognosis of this process is unfavorable, because restitutio ad integrum is impossible.

The treatment is the same as that of the primary disease.

FIG. 73.

Distribution of ascending secondary degeneration a, cervical cord; b–d, dorsal cord. Site of compression of the cord at e; f, lumbar cord below the site of compression with secondary descending degeneration. Natural size.

b. Ascending secondary degeneration of the columns of the spinal cord occurs as the result of disease of the posterior nerve roots or spinal ganglia. Senger produced it experimentally in dogs by section of the posterior nerve roots; in man it is observed most distinctly in disease of the cauda equina. It is confined to the columns of Goll and to the lateral cerebellar tracts (Fig. 73, *a* to *d*).

In the columns of Goll it may be traced as far as the restiform bodies of the medulla, in the cerebellar tracts it extends to the cerebellum. On account of the primary affection, this degeneration is usually bilateral. The degeneration of the columns of Goll is assumed to occur when they are separated from their trophic centres—the spinal ganglia. Some authors regard the columns of Clark as the trophic centres of the cerebellar tracts, but whether these tracts contain centripetal or centrifugal fibres is unknown. Ascending degeneration gives rise to no clinical symptoms.

c. Combined secondary degeneration of the columns of the spinal cord occurs when the continuity of the cord is interrupted entirely or in part. In the former event, for example, in compression of the cord, the columns of Goll and the cerebellar tracts degenerate above the primary disease (ascending degeneration), the decussated and non-decussated pyramid tracts degenerate below the focus of disease. But the cerebellar tracts are only affected if the primary disease is situated in the dorsal or cervical region, since their supposed trophic centre, the column of Clark, begins in the middle of the dorsal region. The secondary degeneration does not begin in the immediate vicinity of the primary disease. The latter generally presents a diffuse change of the entire section of the cord, then follows, in an upward direction, degeneration of the entire posterior columns and, finally, of the columns of Goll alone (Fig. 73, *c*

and d). As a matter of course, the degeneration is bilateral, unless one-half of the cord alone is diseased. Secondary degenerations sometimes follow circumscribed affections of the cord.

3. Amyotrophic Lateral Sclerosis.

I. ETIOLOGY.—Charcot applied this term to a disease which is, to a certain extent, a combination of primary degeneration of the pyramid-lateral tracts and of the large ganglion cells in the anterior horns and in the nuclei of the medulla, particularly the hypoglossus, pneumogastric, spinal accessory, and facial.

FIG. 74.

Cold and exposure to wet are generally mentioned as the causes of the disease. It is most frequent from the ages of twenty-five to fifty years; women are affected more often than men. Weir Mitchell reported a case which was apparently due to working in lead. Seelig-mueller described four cases in brothers and sisters whose parents were cousins. In these cases the disease began in infancy.

II. ANATOMICAL CHANGES.—According to Charcot, the disease begins in the lateral columns of the cord (pyramid tract). It is most extensive in the cervical cord, and gradually diminishes towards the lumbar region (Fig. 74). Inferiorly, the degenerated parts gradually approach the pia mater; superiorly, it may be traced to the lower part of the pons (as far as the pyramid tracts are collected into a bundle), and even into the foot of the peduncle. The internal capsule almost always escapes.

In some cases the undecussated portion of the pyramid tract has been found to be diseased.

Changes in amyotrophic lateral sclerosis. After Charcot. *a*, medulla oblongata; *b*, cervical cord; *c*, dorsal cord; *d*, lumbar cord. The diseased parts are darkly shaded. Natural size.

According to Charcot, the disease is a primary inflammation of the nerve fibres, which gives rise secondarily to an affection of the interstitial connective tissue. The microscope reveals proliferation of the interstitial tissue, destruction of nerve fibres, thickening of the vessels, granulo-fatty cells, and amyloid bodies. Perhaps it is more proper to regard the disease as an atrophy of the nerve fibres and ganglion cells.

Through the medium of the diseased nerve fibres, the disease is said to extend secondarily to the gray matter of the anterior horns; the neuroglia then proliferates, the ganglion cells atrophy and disappear. These changes are most marked in the cervical region, gradually diminish in a downward direction, and are generally absent in the lumbar cord.

Superiorly, they extend to the ganglion cells of the nuclei in the me-
dulla, particularly of the hypoglossus, pneumogastric, spinal accessory,
and facial.

Atrophic and degenerative changes are found in the anterior spinal
roots and the peripheral nerves. The corresponding muscles are atro-
phic, the interstitial connective tissue is increased and often contains an
unusual amount of adipose tissue, so that the muscle may appear even
larger than normal.

Leyden showed that the mode of development is not always as regular as in
the description just given, but that the disease sometimes begins in the gray
matter, and then extends to the white substance. In addition, he showed that
the posterior columns are sometimes affected at the same time. Fig. 75, which
is taken from Leyden's work, shows gray degeneration of the columns of Goll.
Weiss reported a case in which the ganglion cells of the posterior horns had un-
dergone atrophy, and attributes to this lesion the decubitus which developed upon
the trochanters.

III. SYMPTOMS.—The clinical history is a combination of that of

FIG. 75.

Changes in amyotrophic lateral sclerosis with implication of the columns of Goll. Cervical enlarge-
ment. Diseased parts are shaded. After Leyden. Enlarged 5 times.

spastic spinal paralysis and of progressive muscular atrophy and bulbar
paralysis. Sensation and the bladder and rectum are unaffected. The
scene generally opens with paralysis, rigidity, spasms and contractures
of the muscles, then rapid emaciation. As a rule, the symptoms begin
in the upper limbs and extend downwards, but atrophic changes do
not occur in the lower limbs. Atrophy and paralysis follow in the dis-
tribution of the hypoglossus, vagus, spinal accessory, and facial.

More rarely, the symptoms begin in the lower limbs or in the distribution of
the bulbar nerves.

The first symptoms in the upper limbs occur with or without pro-
dromata (formication and other paræsthesiæ). The limbs feel weak and
well-marked paralysis gradually develops, followed by stiffness, spasms
and contractures. The arms are drawn against the trunk, and motion
at the shoulder-joint is attended with pain; the forearms are semi-flexed
and at the same time pronated, and there is generally contracture at the

wrist-joint in a position of pronation, with the fingers drawn into the palm of the hand (Fig. 76).

These symptoms are the result of disease of the pyramid tracts. The affection of the gray matter is shown by rapid atrophy of the muscles of the arms, affecting large masses at one time. Fibrillary contractions are observed during the emaciation. The atrophic muscles are tender on pressure, and generally show the partial degeneration reaction.

If the process extends to the trunk and lower limbs, the patients complain of stiffness in the back, and are hindered in sitting up, and moving the head; they are compelled finally to keep to bed. The gait peculiar to spastic spinal paralysis is produced as the result of muscular rigidity and contracture, and the tendon reflexes are exaggerated. As a rule, the lower limbs do not undergo atrophy.

Bulbar symptoms begin with atrophy and fibrillary contractions in

Fig. 76.
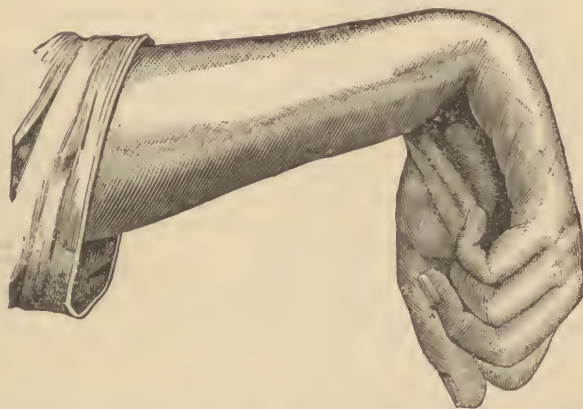
Position of the forearm and hand in amyotrophic lateral sclerosis. After Charcot.

the tongue, and interference with articulation. Paralysis of the palate then gives rise to difficulty in deglutition, and speech becomes indistinct and nasal. The lips atrophy, the mouth is half opened, and continually discharges saliva. The expression of the face is tearful, and, while the forehead appears wrinkled, the lower half of the face is devoid of expression. This condition often leads to "foreign-body" pneumonia or attacks of suffocation and death. Charcot regards the disease as absolutely fatal in one to three years, but Weir Mitchell observed recovery under the use of potassium iodide and sulphur baths.

IV. DIAGNOSIS.—The disease is distinguished from progressive muscular atrophy by the fact that in the latter the paralysis and atrophy go hand in hand, the atrophy is fascicular and generally begins in the interossei, the thenar and hypothenar eminence; muscular rigidity does not occur, and the tendon reflexes are lost.

It is distinguished from hypertrophic cervical pachymeningitis by the predominance, in the latter, of irritative symptoms at the outset, the development of severe sensory disturbances, non-extension to the bulbar

nuclei, and the possibility of recovery. The treatment is similar to that of spastic spinal paralysis.

c. TRAUMATIC AFFECTIONS OF THE SPINAL CORD.

1. *Unilateral Lesions of the Spinal Cord.*

(*Brown-Séquard's Paralysis.*)

I. ETIOLOGY.—Cases in which one-half the transverse section of the cord is rendered incapable of function present characteristic clinical symptoms, independent of the etiology of each individual case.

It occurs most frequently as the result of wounds with a knife or dagger, which have injured only one-half the cord. This happens more rarely in fractures, luxations, exostoses, or neoplasms of the vertebræ. An unilateral lesion may also be produced by exudations, hemorrhages or tumors of the meninges, myelitic foci, sclerosis, hemorrhages and neoplasms of the cord. Rosenthal and Paoluzzi observed each a case following a cold.

II. SYMPTOMS.—The characteristic feature of the symptomatology of an unilateral lesion of the cord is the occurrence of motor paralysis on the same side and sensory paralysis on the opposite side. If the disease is situated high up, we will find motor paralysis of the arm and leg on the injured side, and loss of sensation on the opposite half of the body exactly to the median line and to the level of the site of disease. If the morbid process affects the dorsal cord, motion is paralyzed in one lower limb, sensation in the other lower limb.

These symptoms may develop suddenly as the result of injury, often slowly as the result of other causes. They may lose in distinctness if the spinal affection extends to the other side of the cord.

Upon the side of the body corresponding to the injured side of the cord, there is complete or almost complete paralysis of all the muscles whose nerves leave the cord below the site of injury. The thoracic and abdominal muscles are affected, according to the level of the disease.

At the same time, hyperæsthesia is noticeable in the paralyzed parts. As a rule, this affects all modes of sensation, more rarely the hyperæsthesia is partial, *i. e.*, it affects only certain qualities of sensation. The muscular sense is sometimes retained, sometimes absent.

The upper border of the hyperæsthetic region is formed by a narrow anæsthetic zone (Fig. 77, *b*). This is the result of the direct injury of the posterior nerve-roots. Above this is another hyperæsthetic zone which extends beyond the median line to the other side of the body (Fig. 77, *c* and *c'*).

The symptoms mentioned are associated with vaso-motor disturbances, viz., redness and increased heat of the skin. If the injury affected the cervical cord high up, there will be unilateral symptoms of paralysis of the sympathetic on the same side: redness and increased heat of the side of the face and concha of the ear, contraction of the pupil, narrowing of the palpebral fissure, injection of the conjunctiva, increased flow of tears; hemicrania has also been noticed.

Trophic disturbances are often though not constantly observed; they depend probably on implication of the nerve-roots. Acute decubitus has been observed; sometimes the paralyzed muscles manifest loss of

faradic excitability. Alessandrini mentions pain in the joints on the paralyzed side, the result of extravasations of blood into the joints.

After a while, the paralyzed muscles undergo atrophy from disuse, associated with diminution of electrical irritability.

The cutaneous and tendon reflexes have been found increased in a number of cases.

Paralysis of the bladder and rectum is frequently described. Fischer observed erection of the penis; sexual power is generally abolished.

As a rule, there is complete, more rarely partial, anæsthesia on the side of the body opposite to the site of the lesion; the muscular sense is alone retained. Motor power is almost always intact on this side, but

FIG. 77.
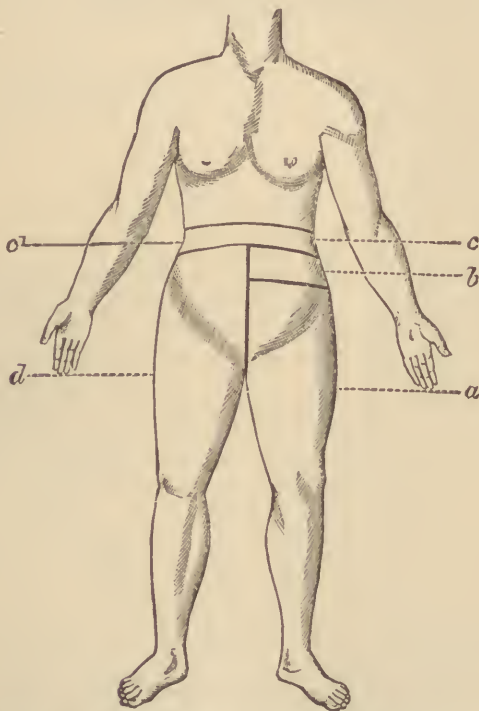
Schematic representation of the symptoms in a unilateral lesion of the left side of the dorsal cord. a, Paralysis of motion and vaso-motor nerves, hyperæsthesia; b, anæsthetic zone (from injury to the sensory nerve roots); c, upper hyperæsthetic zone; c', the same on the uninjured side; d, anæsthesia.

slight paresis is occasionally noticed. The reflexes are unchanged, and vaso-motor disturbances are absent; in rare cases the temperature of this side of the body is elevated.

Above the region of anæsthesia is a narrow zone of hyperæsthesia, continuous with that on the opposite side of the body (Fig. 77, c').

The patients often complain of a cincture feeling, which corresponds to the level of the lesion. Pain and paræsthesiæ may occur upon one or the other side of the body.

If the cause permits, the symptoms are capable of recovery. In such

cases, according to Brown-Séquard, the motor functions recover earlier than the sensory functions, in contradistinction to what happens after the interruption of conduction in peripheral nerves. In other cases the symptoms persist, or cystitis and decomposition of urine set in and the patients die, or finally the morbid process spreads and the symptoms of a diffuse lesion of the cord make their appearance.

The symptoms of unilateral lesion of the cord are most sharply defined if the dorsal cord is affected. In affections of the cervical enlargement, the paralysis in the upper limbs is often confined to certain groups of muscles, because the nerve-tracts for the individual muscles are relatively far removed from one another in this locality. The cutaneous nerves are here given off in such a manner that the hyperæsthetic and anæsthetic zones are not circular but irregular in shape.

III. DIAGNOSIS, PROGNOSIS, AND TREATMENT.—The diagnosis is
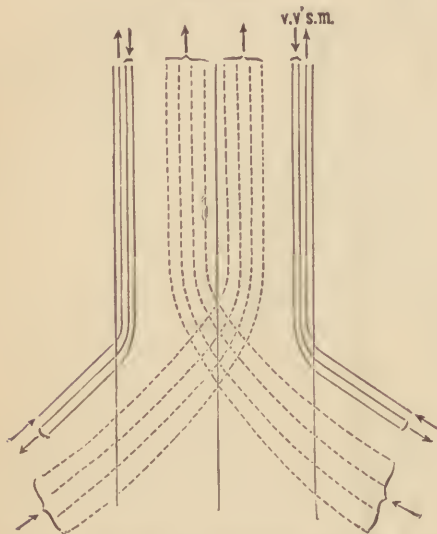
FIG. 78.   FIG. 79.
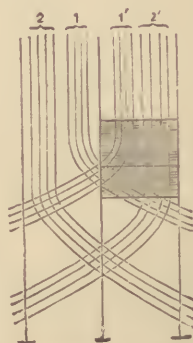
FIG. 78.—Schematic representation of the course of the fibres in the cord. After Brown-Séquard. v, uncrossed motor fibres ; v', uncrossed vaso-motor fibres ; sm, uncrossed fibres of muscular sense ; s, decussating sensory fibres.

FIG. 79.—Schematic representation of the anæsthetic zone on the side of the lesion. The (shaded) focus of disease has interrupted all the sensory fibres on the left side which have entered the cord lower down, while it interrupts only a few of the sensory fibres on the right side. After Brown-Séquard.

easy, since in cerebral processes the paralysis and anæsthesia are always on the same side, and cerebral nerves are also affected. In hysterical hemianæsthesia the special senses are also anæsthetic.

The prognosis and treatment depend on the primary disease.

IV. *Pathologico-physiological Remarks.* The motor nerve-fibres pass from the cerebral cortex to the internal capsule and the foot of the cerebral peduncle, thence through the pons, and, after the decussation in the medulla oblongata, pass in great part into the opposite side of the spinal cord. Here they pass along the posterior part of the lateral column (Fig. 70, 3 *psb*), then into the gray matter and the anterior nerve roots of the same side. Hence a unilateral lesion of the cord must produce paralysis of all the motor nerves on the same side which are situated below the lesion.

But it must be remembered that the decussation of the motor fibres in the medulla is not complete, a small portion remaining upon the same side of the cord. This is the pyramid-anterior column tract, which is adjacent to the anterior longitudinal fissure (Fig. 70, 1 *pvs*). This tract decussates within the spinal cord, and passes to the other side. Hence, a slight degree of motor paralysis may develop upon the side opposite to the spinal lesion. Flechsig has shown that, in some cases, the undecussated pyramid tract may be the larger, so that, in exceptional instances, the paralysis may be situated on the side opposite to the spinal lesion.

The sensory fibres must be assumed to decussate within the spinal cord, since this alone will explain the crossed anæsthesia in unilateral lesion of the cord (vide Fig. 78).

The fibres for muscular sense and the vaso-motor fibres do not decussate in the cord, since they are paralyzed on the side of the lesion.

According to Brown-Séquard, the decussation of sensory fibres of the different qualities of sensation occurs at different levels in the cord; going from below upwards the fibres for tactile sensation first decussate, then those for tickling, pain, and temperature.

The hyperæsthesia on the side of the lesion is explained by some as the result of irritation of the sensory nerve fibres by the wound, by others as the result of paralysis of inhibitory mechanisms.

For an explanation of the narrow anæsthetic zone on the paralyzed side, we refer to Fig. 79.

2. *Acute Injuries of the Spinal Cord.*

I. ETIOLOGY.—As a rule, acute injuries of the spinal cord are associated with external traumata. In some cases, however, severe lesions of the spinal cord are produced by an injury, although nothing is noticeable in the spine or soft parts. Moreover, sharp weapons may penetrate the intervertebral spaces and injure the cord without injuring the bony parts of the spine.

Acute injuries of the cord are relatively frequent in fractures and dislocations of the vertebræ. Detached fragments of the bone may enter the cord, or the dislocation narrows the spinal canal and compresses the cord. In certain cases this is preceded by disease of the vertebræ, most frequently tuberculosis. In such an affection of the atlas and axis, a sudden movement of the head may dislocate the odontoid process and compress the cord. Or the carious vertebræ sink in and produce such marked flexion and narrowing of the spinal canal as to injure the cord. Charcot observed rupture of the cord in the new-born as the result of excessive traction during delivery.

II. ANATOMICAL CHANGES.—If death occurs soon after a simple incised or stab wound, we will find a gaping wound in the cord which is generally filled with blood clots. The substance of the cord soon undergoes swelling and softening at the cut ends, and the microscope shows fatty degeneration of the tissue elements. This terminates in the formation of a connective tissue which is rich in cells, which continues to proliferate and thus restores the continuity of the cord by a sort of cicatricial tissue. Whether the regeneration of nerve fibres occurs in man is unknown, though clinical experience favors the belief in such an occurrence.

The wound in the cord sometimes contains a part of the weapon, and under such circumstances an abscess is apt to develop.

If the patient survives the injury for a long time, ascending and descending secondary degeneration will develop.

In other cases, the cord undergoes contusion, hemorrhagic disintegration, or rupture. Fatty degeneration, softening, and secondary degener-

ations follow. The meninges may take part in the changes, or remain entirely intact.

III. SYMPTOMS.—As a rule, there is paraplegia of motion and sensation, paralysis of the bladder and rectum, and changes of reflex excitability.   Vaso-motor symptoms (changes in the color and temperature of the skin, and in the formation of perspiration) have not infrequently been described. Priapism is a frequent symptom; the penis is generally semi-erect or flaccid. This is observed most commonly in injury of the cervical or dorsal cord; it is absent if the lesion is situated below the third lumbar vertebra. All these symptoms occur very soon after the injury.

In injury of the cord above the cervical enlargement, death may occur very rapidly. If life is spared, all the limbs are paralyzed as regards motion and sensation; reflex irritability is sometimes diminished or abolished immediately after the injury, but is increased after the cord has, to a certain extent, recovered its tone. Retention of urine and fæces is often observed at the start, but this disappears after a time, and may be followed by paralysis of the bladder and rectum (cystitis, ammoniæmia, decubitus). Paralysis of the abdominal and thoracic muscles interferes with respiration; if the diaphragm is paralyzed, death from suffocation will probably result in a short time. Implication of the medulla oblongata will be shown by difficulty in deglutition, vomiting, slowness of the pulse, pupillary changes. Abnormally high temperature of the body has been observed in several cases. According to the experiments of Nannyn and Quincke, there are certain cerebro-spinal fibres which inhibit the production of animal heat.

Injury to the cervical enlargement is also followed by motor and sensory paraplegia of all the limbs. The reflexes, the bladder, and rectum present the same changes as have been described above.   Respiration is impeded on account of paralysis of the thoracic and abdominal muscles. The cerebral nerves are unaffected, at the most there are pupillary changes (cilio-spinal centre in the cervical cord).

In injury to the dorsal cord, the motor, sensory, and vaso-motor disturbances are confined to the lower limbs; bladder and rectum as described above. The situation of the injury can be determined by accurately localizing the upper boundary of anæsthesia. The anæsthetic region is sometimes bounded by an apparently hyperæsthetic zone.   In Nieden's case of injury of the cord at the level of the first dorsal vertebra, the temperature in the rectum, on the day of admission to the hospital, was only 35.1° C., on the following day (day of death) only 27.5° C.

In injury to the lumbar cord, there is paraplegia of the lower limbs, permanent paralysis of the bladder and rectum, and abolition of reflex irritability.   The paralyzed muscles often present rapid atrophy and loss of faradic excitability.

The disease may last for weeks, months, or years. But, as a rule, death soon occurs as a direct result of the spinal lesion. It often follows at a later period as the result of increasing marasmus, paralysis of the bladder, and decubitus. Recovery is rarely observed, and only after partial or comparatively slight injuries.

IV. DIAGNOSIS, PROGNOSIS, AND TREATMENT.—The diagnosis is easy, when motor and sensory paraplegia follows an injury to the spinal column or its vicinity. The prognosis is almost always unfavorable, and recovery can hardly ever be expected.

The treatment depends upon the causal conditions. Opinions are

divided with regard to the advisability of trephining. In other respects the treatment is purely symptomatic : absorbents (iodine, mercury), careful attention to the bladder and rectum, prevention of bed sores, constant current.

3. *Concussion of the Spinal Cord.*

(*Railway Paralysis.*)

I. ETIOLOGY.—Concussion of the spinal cord is the term applied to those diseases of the cord which are the result of violent mechanical injury of the entire body, or chiefly of the spinal column, without injury to the vertebræ or meninges. This may occur after a fall or blow upon the spine or buttocks, feet, or arms. Special attention has been paid recently to concussion after railway accidents (railway spine), in which the severity of the symptoms is often entirely disproportionate to the antecedent lesion. Rigler suggests that this is owing to the fact that fright aids materially in producing the spinal symptoms. Locomotive engineers and brakemen sometimes suffer from spinal symptoms, although they have suffered no gross concussion as in railway accidents.

II. ANATOMICAL CHANGES.—Anatomical changes may be wanting, although death occurs shortly after the accident. This was observed by Leyden, in a case which proved fatal in five days, and by Fischer in a case which ended fatally in two days. We must then assume purely molecular changes in the substance of the cord.

In other cases hemorrhages are found in the cord and its meninges. If they are numerous and extensive, they may be followed by inflammation and softening processes in the cord.

In still other cases, the symptoms of chronic meningitis, myelitis, or myelo-meningitis gradually develop. In a case of this kind, in which death occurred three years after a railway accident, Leyden found a cheesy, tubercular inflammation in the peridural cellular tissue, and acute myelitis in the cervical enlargement, with ascending and descending secondary degeneration. The inflammation had also passed through the intervertebral foramina, into the left brachial plexus.

The development of tumors, especially gliomata, is also mentioned as a sequel of spinal concussion.

III. SYMPTOMS.—The symptoms are extremely variable. In one series of cases the accident is immediately followed by the signs of shock (small pulse, accelerated breathing, cool and cyanotic skin, paralyses, involuntary evacuations, etc.) which terminates fatally in a few hours or days.

In other cases, the symptoms of collapse are less marked and gradually disappear, the paralyses gradually improve, and complete recovery occurs at the end of days or weeks.

In a third group of cases, recovery may be delayed for years, but finally becomes complete.

Of great practical importance are those cases in which no morbid phenomena are observed immediately after the accident, and the symptoms of a chronic affection of the cord or meninges do not develop until after the lapse of weeks, even of months.

Sometimes meningeal, *i. e.*, irritative symptoms predominate, sometimes spinal symptoms, *i. e.*, paralyses. In some cases the symptoms resemble those of multiple spinal sclerosis (Westphal), sometimes those

of spastic spinal paralysis, etc. Such cases, as a rule, terminate in death.

If the brain has also suffered concussion, the symptoms of the latter generally obscure the spinal phenomena. In other patients, cerebral symptoms (mania, progressive paralysis of the insane, etc.) develop at a late period.

In four cases of railway spine, Wharton Jones observed amblyopia as the result of atrophy of the optic nerve.

IV. DIAGNOSIS.—The diagnosis is easy if there is an undoubted relation between the spinal symptoms and a previous concussion of the body or spinal column. In guarding against simulation after railway accidents, Schulz recommends attention to the tendon reflexes which, he claims, are always changed, sometimes exaggerated, sometimes diminished.

V. PROGNOSIS.—The prognosis is always serious. The symptoms sometimes grow unexpectedly worse, or they do not appear until a long time after the accident. The prognosis is especially grave when we are justified in assuming serious changes in the substance of the cord.

VI. TREATMENT.—If the symptoms of shock predominate, we should order stimulant measures: friction of the skin, coffee, brandy, camphor, musk, ammonia, etc.

If the disease runs a chronic course, we may order absorbents, particularly potassium iodide, iodine and iron baths, and electricity.

In other respects, purely symptomatic treatment.

4. *Slow Compression of the Spinal Cord.*

(*Compression Myelitis.*)

I. ETIOLOGY.—The causes of compression sometimes enter the spinal canal through the intervertebral foramina; they start from diseases of the vertebræ themselves, from changes in the peridural cellular tissue and meninges, or from diseases of the spinal cord itself.

Among lesions of the first class may be mentioned aneurisms of the abdominal aorta, which have eroded the vertebræ, abdominal echinococci, neoplasms and inflammations which have extended through the intervertebral foramina into the spinal canal.

Among the diseases of the vertebræ, tuberculosis occupies a prominent part. Of rarer occurrence is vertebral cancer, which is generally secondary, more rarely primary. In certain cases, the compression of the cord is the result of syphilitic changes in the bones, osteomata, exostoses, luxation or arthritic changes in the vertebræ, etc. The compression is sometimes the result of thickening of the odontoid process.

Diseases of the meninges and peridural tissue, which may give rise to compression, include inflammatory changes, neoplasms, and parasites. In some cases, the primary affection is situated in the vertebræ, but is followed by changes in the meninges, which exercise pressure on the cord.

Compression of the cord is rarely the result of the diseases of the cord itself. This has been observed in tumors of the cord (glioma, sarcoma, carcinoma, tubercle, gumma) and in cystic dilatation of the central canal.

The changes described above may not alone give rise to compression of the cord, but also to inflammatory processes in the meninges, nerve roots, and the cord itself.

II. ANATOMICAL CHANGES.—So far as regards the mechanical results of compression, the cord will be found flattened and thinned at the site of compression. These appearances are sometimes unilateral, sometimes they extend around the entire circumference of the cord. The parts immediately above and below the site of compression are sometimes dilated into a spindle shape (vide Figs. 80 and 81).

These mechanical effects are not the chief factors in the clinical symptoms, but the latter are the result of the inflammatory changes which often affect the nerve roots, meninges, or the cord itself.

In the majority of cases, the cord is, indeed, thin and flattened, but at the same time it is soft, diffluent, sometimes very pale and transparent, sometimes injected, i. e., it presents the appearances of transverse myelitis. If the changes have existed for some time, the affected

FIG. 80.　　　　FIG. 81.
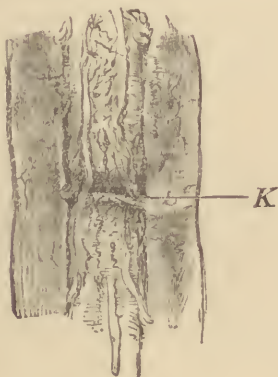
Anterior surface of cord.　　　Posterior surface. The dura mater opened and turned back.

Annular compression and narrowing of the cord (at k) as the result of cancer of the vertebræ in a woman æt. 34 years. Natural size. Complete interruption to conduction during life.

part of the cord not infrequently becomes very firm and sclerotic, and ascending and descending secondary degeneration develops.

The lesions are chiefly of an interstitial character; the cellular elements of the neuroglia increase in number and size, and the nuclei also increase in number; the basement substance becomes more abundant. The blood-vessels present thickening of their walls, increase of the nuclei, and fatty degeneration. The medullary sheath of the nerve fibres undergoes granular disintegration and gradually disappears; the axis cylinders are swollen, and contain vacuolæ; later they are destroyed. Distention, increase of the nuclei and pigment, vacuolæ, and atrophic changes have been observed in the ganglion cells.

The nerve roots and intervertebral ganglia also present inflammatory and atrophic changes, the former constituting the initial, the latter the terminal stage.

The meninges at the site of compression are either markedly congested or unusually pale. They are often thickened, opaque, and adherent to one another. These changes may play a prominent part in the history of compression-myelitis. This is particularly true of the

majority of cases following vertebral caries, since compression of the cord is rarely the result of flexion of the spinal canal, but rather of inflammation and thickening of the outer surface of the dura mater (external pachymeningitis). In rare cases of vertebral caries, pus enters the peridural space and compresses the spinal cord.

III. SYMPTOMS.—Compression-myelitis may be divided into the prodromal, irritative, and paralytic stages. The symptoms of the prodromal stage depend upon the causes of the disease in each individual case. The irritative stage is characterized by symptoms which indicate irritation of the meninges, nerve roots, and cord. The symptoms of the paralytic stage depend upon compression and inflammation of the spinal cord.

These stages are not always sharply defined. When the compression is the result of intra-medullary diseases, the symptoms generally begin with paralytic phenomena.

We cannot enter into a detailed description of the prodromal phenomena; the chief ones are pain and tenderness in certain parts of the spine, pain during movement, and curvatures of the spine.

Inflammatory irritation, particularly in the nerve-roots, is shown by neuralgiform pains. These radiate into the limbs, or pass around the trunk (cincture feeling) ; they are sometimes localized in definite parts, and may be especially severe at night. They are sometimes increased on movement and concussion of the spine.

Hyperæsthesia and paræsthesiæ are often noticed in the distribution of the affected nerves. Charcot has also observed dysæsthesia (vide page 86). Trophic disturbances (herpes zoster, bullous eruptions, acute decubitus, joint changes, etc.) have been observed not infrequently. The patients sometimes complain of twitchings and contractures in the muscles.

Paralysis slowly develops and gradually occupies the foreground. It occasionally develops very rapidly, almost in an apoplectiform manner. In compression of the cervical cord, it sometimes happens that the arms alone are paralyzed.

Previous hyperæsthesia gradually gives way to increasing anæsthesia, although complete anæsthesia is rare. In many cases, sensation is very little affected, despite the severe motor symptoms. Delayed conduction is sometimes noticeable. In addition to anæsthesia, the patient may suffer from the most violent pains (anæsthesia dolorosa), particularly in cancer of the vertebræ. If these symptoms are the result of compression of individual nerve-roots, they will be confined to circumscribed areas, and associated with loss of reflex excitability in the affected nerve-tracts. The affected muscles then undergo rapid atrophy, and degeneration reaction develops. But if the symptoms result from compression of the spinal cord itself, the motor and sensory disturbances almost always assume a paraplegic character, and the cutaneous and tendon reflexes are considerably increased. The signs of spinal hemiplegia occur in very rare cases as the result of unilateral compression of the cord. Reflex excitability is abolished if the lumbar cord is compressed, or if extensive changes in the gray matter have extended from a focus situated higher up. The paralyzed limbs sometimes present vaso-motor symptoms : discoloration of the skin, elevation of temperature, and abnormalities in the production of perspiration. Kahler and Pick described a case in which the motor irritation began with ataxic symptoms. Disturbances

of the bladder and rectum occur from the start in diseases of the lumbar cord.   Under other conditions, they may remain long unaffected.

Complete or almost complete recovery may take place, even if the paralysis is very marked.   Improvement often occurs with surprising rapidity. I know of a case of complete paraplegia of three or four years' duration, following vertebral caries, in which, at the end of that time, improvement occurred with such rapidity that the patient was able, at the end of two weeks from the first signs of recovery, to walk unaided to the dining-room.   In such cases, we may not assume acute regeneration of compressed and destroyed nerve elements.   It must be assumed that the few intact nerve fibres at the site of compression suffice, when the compression ceases, to conduct impressions.

Some cases present repeated exacerbations and remissions.   In others, the disease progresses uninterruptedly.   Increasing marasmus, decubitus, cystitis, and ammoniæmia, more rarely sudden bulbar paralyses, cause the fatal termination.

Secondary degeneration is shown by the fact that the previously flaccid paralyzed muscles become stiff, and twitchings and contractures make their appearance.   In the lower limbs, the extensors are at first chiefly affected, but later the flexors becomes involved to a more marked extent—the thighs are strongly flexed and drawn upwards, and the legs are flexed so that the heels come in contact with the thighs.   Secondary degenerations may also give rise to recurrent paralyses, *i. e.*, paralysis of the upper limbs becomes associated with paralysis of the lower limbs, which resulted from a morbid process in the lumbar or dorsal cord. This must be explained on the assumption that the secondary degeneration has interrupted the paths of conduction to the upper limbs.

IV. DIAGNOSIS.—While the diagnosis of compression of the cord is usually easy, that of the cause of each individual case may be difficult. This is especially true if no abnormality can be discovered in the spinal column, and if the previous history furnishes no clue.

V. PROGNOSIS.—This depends upon the etiology, and, as a matter of course, it is unfavorable when the cause cannot be removed.   Otherwise, improvement and even complete recovery are not infrequent, particularly in vertebral caries.

VI. TREATMENT.—This must first be directed towards the removal of the cause.   In other respects, the treatment is similar to that of acute and chronic myelitis (see page 88).

B.—FUNCTIONAL DISEASES OF THE SPINAL CORD, OR SPINAL NEUROSES.

1. *Spinal Irritation.*

1. The term spinal irritation is applied to a symptom-complex characterized by pain in the spine, associated with neuralgiform pains in the skin or viscera, vasomotor and secretory disturbances, motor weakness, functional cerebral changes, etc., and not dependent on an anatomical lesion of the cord.

The condition has been attributed to spinal anæmia or hyperæmia, meningitic irritation, or changes in the sympathetic.   Some writers deny the very existence of spinal irritation.

2. This condition is observed most frequently in women, and is often associated with hysteria, chlorosis, and anæmia.   Bodily and mental strain, exhausting diseases, excesses in venery, abuse of alcohol or opium, and heredity have been mentioned as causes.   It occurs generally from the age of 15 to 30 years.

3. The most constant symptom is pain in the spinal column. It most frequently affects the dorsal spine, but is also observed in other parts. It occurs spontaneously, but becomes especially severe on pressure upon the spinous processes, and may be so violent as to give rise to syncope or epileptiform seizures. It also becomes evident on passing over the spine a sponge dipped in hot water, or the cathode of the galvanic current. The adjacent integument of the spine is not infrequently very hyperæsthetic. Hammond also describes a deeper pain which is produced by percussion of the spine.

In addition, there are eccentric symptoms of irritation which vary according to the affected portion of the cord. In changes in the cervical cord, we notice headache, vertigo, visual disturbances, tinnitus aurium, hyperæsthesia of the face, depression, insomnia; in other cases, somnolence, neuralgiform pains in the face, neck, upper limbs; nausea and vomiting. The eccentric dorsal symptoms of irritation include palpitation, asthmatic attacks, nervous cough, intercostal neuralgia, gastralgia, vomiting, singultus, etc. Lumbar irritation is characterized by pains in the back, lower limbs, bladder, rectum, uterus and ovaries, strangury, polyuria, etc.

Paræsthesiæ, muscular pareses, spasms, and vaso-motor changes are not uncommon.

The disease may last months and years, but hardly ever leads to more serious affections.

4. The diagnosis is usually easy after prolonged observation.

5. The treatment consists of bodily and mental rest, nutritious and abundant food, the administration of quinine, iron, cod-liver oil, cold-water treatment, a trip to the country, and the use of the galvanic spinal current (descending, not too strong or prolonged, anode or cathode to unusually painful vertebræ). The faradic brush to the spine, or galvanization transversely through the skull, has also proved useful. Strychnine, potassium bromide, zinc, arsenic, phosphorus, and opium have also been recommended.

2. *Functional Weakness of the Spinal Cord. Spinal Neurasthenia.*

1. The etiology of spinal neurasthenia is the same as that of spinal irritation, but the two conditions are not identical. Spinal neurasthenia is especially frequent in men, particularly among the higher classes.

2. The symptoms consist of abnormal functional irritability and weakness of the spinal cord. It is often associated with cerebral neurasthenia. It is distinguished from spinal irritation mainly by the fact that the motor disturbances predominate, while sensory symptoms may be entirely absent.

The patients are easily tired on walking or standing. They often experience that feeling of muscular exhaustion which is felt by healthy individuals only after unusual exertion. This is often felt immediately after waking. The gait is stiff, trembling, and uncertain. Similar symptoms may develop in the upper limbs (while writing, etc.).

Sleep is restless and disturbed by dreams. The patients become depressed, irritable, and shy. Many complain of vertigo, a feeling of pressure in the head, ringing in the ears; in company, they suffer from a feeling of terror and palpitation; in theatre, they have a dread of fire, etc.

Sexual excitement is often noticeable, but its gratification is attended not infrequently with premature ejaculation of semen, and may be followed by syncope (in one of my cases by palpitation and epileptiform seizures).

Anorexia, eructations, flatulence, and constipation are observed not infrequently.

Some patients complain of paræsthesiæ, vaso-motor disturbances (coldness of the hands and feet), hyperæsthesia of the back.

The disease is capable of recovery, often not until after the lapse of months and even years.

3. The treatment is similar to that of spinal irritation. Erb prefers the ascending galvanic spinal currents. Good effects have been obtained from general faradization: the patient is placed in a bath-tub filled with lukewarm water, in which is put the cathode of the secondary current, or the feet are placed upon a large cathode, while a large anode is gradually passed over the different parts of the body from above downwards. Or the physician takes the anode in one moistened) hand, while the patient's body is stroked with the other (also moistened) hand. Duration of each sitting ten minutes, every two to three days.

3. *Acute Ascending Spinal Paralysis.*

(*Kussmaul-Landry's Paralysis.*)

I. ETIOLOGY.—The symptoms of this disease consist of progressive paralysis from below upwards, with retention of electrical irritability in the paralyzed muscles, absence of atrophy, of sensory and trophic disturbances, of paralysis of bladder and rectum, and of anatomical changes in the nervous system.

The disease is rare, but is much more common in men than in women, and, as a rule, appears between the ages of twenty and forty years.

The causes are often unknown. It has been attributed to cold, suppression of menstruation, and mental excitement. Bablon observed it after coitus in the standing position. It is relatively frequent after acute infectious diseases, and has also been attributed to syphilis.

II. SYMPTOMS.—The paralysis develops unexpectedly, or is preceded by prodromata. The latter consist of chilliness, slight fever, pain in the back and limbs, formication, and other paræsthesiæ.

The motor paralytic symptoms do not always follow a strictly ascending course. They begin in one or both feet, then affect the legs, thighs, loins, abdomen, chest, hands, forearms, and arms, then the neck, and finally the bulbar nerves.

Westphal described a case in which bulbar paralyses were alone observed. In rare cases the paralysis pursues a descending course.

The motor paralysis begins, as a rule, with paresis which gradually deepens into complete paralysis; the limbs remain flaccid.

The patients first experience a tired feeling in the legs, and are soon compelled to take to bed. If the dorsal muscles are affected, they are unable to sit up, and paralysis of the abdominal muscles interferes with defecation, coughing, sneezing, *i. e.*, with all expiratory processes. Paralysis of the intercostal muscles impedes inspiration. Gradually the movements of the hands, forearms, and arms are impaired, then the movements of the head are interfered with. Phrenic paralysis will produce the most marked dyspnœa.

Then bulbar symptoms gradually make their appearance. Speech becomes difficult on account of paralysis of the hypoglossus. Paralysis of the palate and œsophagus makes the speech nasal, and produces difficulty in deglutition. The pulse becomes very much accelerated, and attacks of suffocation occur which terminate fatally. As a rule, the cerebral nerves remain unaffected.

The paralyzed muscles retain their electrical irritability to the last, and do not undergo atrophy.

Cutaneous sensibility is very little, if at all, affected. Anæsthesia and hyperæsthesia have been observed in a few cases. Delayed conduction of the sensations of pain and temperature has also been reported. Diminution of muscular sense has also been noticed.

Decubitus and other trophic disturbances do not occur.

The cutaneous and tendon reflexes are unchanged at first, but gradually they are diminished and finally abolished.

The bladder and rectum are not paralyzed, although temporary retention of urine sometimes occurs at the outset.

The general condition may be unchanged. Febrile movement may

or may not be present. Enlargement of the spleen and albuminuria have been observed in some cases.

The disease runs an acute, progressive course. Death occurs generally in the middle of the second week of the disease, sometimes in two or three days, at others in two or three weeks.

In rare cases recovery ensues even after bulbar symptoms have developed. Several months often elapse before the status quo ante is restored. Remissions and exacerbations, with a finally fatal termination, have also been observed.

III. ANATOMICAL CHANGES.—The nervous system presents no anatomical changes in cases of this disease. But enlargement of the spleen and swelling of the intestinal lymph follicles and mesenteric glands have been observed in a number of cases, and this lends a certain degree of plausibility to Westphal's suggestion that the disease is the result of some unknown infection.

IV. DIAGNOSIS.—The disease is distinguished from ascending myelitis by the absence of fever, sensory disturbances, paralysis of the bladder and rectum, decubitus, and changes in electrical excitability. Acute anterior poliomyelitis does not possess a progressive character, and results in rapid muscular atrophy and loss of electrical excitability. In acute multiple neuritis, sensory disturbances predominate, and the electrical irritability of the affected nerves and muscles rapidly diminishes.

V. PROGNOSIS.—The prognosis is always grave, although recovery is not impossible. The more acute the course, and the more prominent the bulbar symptoms, the earlier will the fatal termination occur.

VI. TREATMENT.—If the disease has been preceded by syphilis, antisyphilitic remedies must be administered.

Other measures recommended are: *a. Externally*—actual cautery, cups, leeches, ice-bag, derivatives to the spine, cold compresses. *b. Internally*—potassium iodide, ergotine, atropine, etc. The galvanic spinal current may also be employed.

4. *Reflex Paralysis.*

1. This term is based on the theory that diseases of the peripheral organs may impair the function of the spinal cord to such an extent as to give rise to spinal paralyses, which almost always appear as paraplegia. The disease is most frequent as the result of affections of the genito-urinary apparatus and intestinal tract; it has also been observed after injury, inflammations of joints, and cold.

Among the diseases of the urinary apparatus, we may mention cystitis, retention of urine, nephritis, and calculi. The morbid changes in the genital tract include gonorrhœa, stricture, phimoses, hypertrophy of the prostate, hydrocele, flexions and ulcerations of the uterus.

Reflex paralysis is also said to follow dysentery, more rarely simple diarrhœa, improper administration of cathartics, constipation, colic, worms.

Paralyses of a spinal character have been observed after gunshot wounds which did not directly involve the paralyzed limbs. They have also been reported as the result of ulcers of the skin and of a cold.

2. Brown-Séquard believes that the paralysis is the result of spinal anæmia due to reflex spasm of the vessels, but it is hardly conceivable that such a spasm may last for years. Jaccoud believes that the peripheral irritation directly inhibits and thus exhausts the functions of the spinal cord. In recent times, however, the view is gaining ground that the majority of the cases are the result of anatomical lesions. As a rule, we have to deal with inflammations which extend from the primarily diseased organ along the peripheral nerves or blood-vessels to the contents of the spinal canal, so that the affection is really the result of ascending or descending degenerative neuritis, meningitic changes, or myelitic processes. But the

possibility of **reflex** paralysis in the strict sense must be granted, particularly when the paralysis disappears almost immediately after its cause is removed.

3. The symptomatology varies according as the disease is the result of neuritis, meningitis, or myelitis. The prognosis depends upon the etiology, and this is also true of treatment.

6. *Psychical Spinal Paralysis.*

(*Fright Paralysis.*)

Paraplegia is sometimes produced by profound emotions. Kohts and Leyden have referred recently to the great influence of fright in the development, not alone of functional, but also of organic diseases of the spinal cord. Their observation is based on the abundant material furnished during the bombardment of Strassburg. Russel Reynolds and Riegel have called attention to the fact that paraplegia may result from the influence of the imagination, particularly in feeble, exhausted individuals. Sometimes there is complete paralysis, in other cases merely paresis; the bladder and rectum are generally intact. Paræsthesiæ and anæsthesia are not infrequent. The treatment is chiefly moral. Iron, quinine, strychnine, electricity, cold frictions, etc., may become necessary.

6. *Paralyses after Acute Diseases.*

(*Infection Paralyses.*)

1. These cases are observed most frequently after acute infectious diseases, particularly after certain epidemics. As a rule, the paralysis develops during convalescence, but it has also been observed at the outset.

The best-known form is that which occurs after diphtheria of the pharynx, other mucous membranes, and skin. It generally occurs in the second or third weeks after recovery, sometimes in the sixth and eighth weeks. It has also been observed after catarrhal angina, and even after herpes pharyngis.

Paralyses sometimes occur after typhoid diseases, particularly after typhoid fever; also after dysentery and cholera. Cases have also been reported after the acute exanthemata (variola, scarlatina, measles, erysipelas, herpes zoster), after erythema nodosum, urticaria febrilis, purpura, puerperal fever; also after pneumonia, whooping-cough, articular rheumatism, pleurisy, malaria.

2. In one series of cases the paralysis is confined to a single nerve, and occasionally even to smaller groups of muscles. In other cases an entire limb is paralyzed, and paraplegic conditions may develop, usually without implication of the bladder and rectum. Or we find the symptoms of acute ataxia, multiple sclerosis, progressive muscular atrophy, spinal atrophic paralysis, acute ascending paralysis, etc. Cerebral hemiplegia with or without aphasia, or the latter affection alone, may develop. It evident that all these conditions cannot be produced by the same anatomical lesions.

The lesions which have been observed are: degenerative neuritis, diffuse myelitis, or disseminated myelitic processes (found after variola by Westphal), multiple sclerosis (also observed after variola), meningitic changes extending to the anterior nerve-roots (in diphtheria), cerebral embolism or thrombosis, etc.

It is now assumed that the majority of cases of paralysis after acute infectious diseases are the result of anatomical lesions, but in rarer cases functional affections of the spinal cord are observed—for example, acute ascending paralysis which presents no anatomical basis. In some instances of unusually rapid disappearance of the paralysis, we must assume conditions of exhaustion of the spinal cord (asthenic paralysis). The intermittent paralysis of malaria must also be regarded as a functional affection. It is characterized by the development of a usually progressive paralysis, with or without anæsthesia, and paralysis of the bladder, which disappears at the end of a few hours after a sweat. The attacks recur at stated intervals, and are cured by anti-malarial remedies.

7. *Toxic Spinal Paralyses.*

More or less severe paralysis may be produced by poisoning with lead, arsenic, phosphorus, mercury, carbonic oxide, alcohol, tobacco, camphor,

copaiba, ergot, absinthe, opium, belladonna, strychnine, etc. The paralyses may be confined to individual groups of muscles, may extend to larger nerve-tracts or to entire limbs, or may be of a hemiplegic or paraplegic character; they may be associated with anæsthesia or hyperæsthesia, or sensation may be unaffected. We will here discuss a few of the most frequent forms of toxic paralysis.

a. Lead Paralysis.

1. Lead paralysis is one of the later symptoms of lead poisoning, and is usually preceded by the formation of a blue line on the gums, more or less frequent colic, and lead arthralgia. Tanquerel des Planches reports one case in which the lead paralysis was produced a week after the patient began working in lead. Usually a number of years elapse before the paralysis is produced. Excessive use of alcohol and strain of certain groups of muscles are said to favor the development of lead paralysis. It is unnecessary to enter here into the various ways in which the system may be poisoned with lead.

2. As a rule, the paralytic symptoms develop very gradually, but more rarely they begin suddenly. Sometimes they follow immediately upon an attack of lead colic.

In the majority of cases, the paralysis is confined to the upper limbs, often only to those muscles which are supplied by the radial nerve. The muscles of the lower limbs are rarely, those of the back still more rarely, affected. Duchenne also observed lead paralysis of the intercostal muscles and diaphragm.

The paralysis generally begins in the right arm (in left-handed persons, in the left arm), but the corresponding muscles of the other arm soon become affected.

The extensor digitorum communis is first affected, and there is difficulty in extension of the first phalanges of the second and third fingers. Then the extensor digit. quint. propr. and extensor indic. propr. become affected; this is followed by paralysis of the extensor carpi rad. et ulnar., next of the extensor pollic. long. et brev.; the abductor pollic long. resists for a long time, and is finally affected with the muscles of the ball of the thumb. The interossei, particularly the first, also become involved.

It is characteristic that the supinator longus et brevis remain intact, unless the biceps and brachialis internus become involved. Paralysis of the latter group is generally preceded by paralysis of the deltoid. The triceps is affected very late or not at all.

In the lower limbs the peronei are first affected, then the extensors of the leg, but the tibialis anticus remains intact.

Degeneration reaction develops in the paralyzed muscles. The direct and indirect faradic irritability of the muscles is gradually abolished, but the paralysis is sometimes more extensive than the disturbances of faradic excitability. If the paralyzed muscles have not lost their faradic irritability, their function is often restored after a few electrical sittings. Direct galvanic stimulation of the muscles shows increased excitability, slow contractions, and predominance of AnClC. The increased galvanic excitability soon disappears, but AnClC remains predominant. Partial degeneration reaction is observed in muscles which are not very markedly affected. If the degeneration reaction is complete, the muscles will not recover, if at all, until the end of three to four months.

The muscles often recover voluntary power, although they are still

inexcitable to the faradic current.   Diplegic contractions have been observed in a number of cases (vide page 127).

During the continuance of the degeneration reaction, the mechanical excitability of the muscles is increased, and they sometimes present fibrillary contractions.   The paralysis is followed by atrophy of the muscles, so that the interosseous spaces appear hollowed out.   A deep groove is seen on the dorsal surface of the forearm, the shoulder is flattened, etc.   Symptoms similar to those of progressive muscular atrophy have been observed in some cases of lead poisoning.

The cutaneous and tendon reflexes are abolished in the regions in which the muscles have lost their electrical excitability.

Trophic disturbances are sometimes noticed : enlargement of the sheaths of the tendons (tenosynitis hypertrophica), enlargement of the phalanges, etc.

The disease runs a chronic course.   Relapses sometimes occur, although the patient was not exposed anew to the influence of lead.

3. The anatomical changes in the muscles consist of narrowing of the fibres, increase of the nuclei of the sarcolemma, gradual disappearance of the muscle substance, proliferation of the interstitial tissue, and sometimes of the adipose tissue.   Some authors regard the muscular changes as primary, the changes in the nervous system as secondary, $i.$ $e.$, as propagated from the muscles.   Others maintain that changes in the peripheral nerves are primary (degenerative atrophy).   In one case Westphal noticed changes in the radial nerve which he attributed to regenerative processes (finest nerve fibres surrounded, in bundles, by a common sheath).

The clinical symptoms point distinctly to an affection of the spinal cord.   They are so like those of subacute and chronic atrophic spinal paralysis that it has been assumed that the lead poisoning first gives rise to a functional disease of the large ganglion cells of the anterior horns of the spinal cord, and this in turn to secondary changes in the peripheral nerves and muscles.

4. The diagnosis is usually easy, not alone from the etiology, but also from the characteristic distribution of the paralysis (radial nerve with intact supinators).   In peripheral radial paralysis, the supinators are affected, as a rule, sensory disturbances are present, and peripheral causes are demonstrable.   The differential diagnosis from spinal atrophic paralysis may be difficult in the absence of a previous history and of other symptoms of lead poisoning.

5. The prognosis is not favorable, as a rule, because the patients generally suffer from relapses unless they give up their occupation. Relapses may also occur without the intervention of renewed poisoning.

In individual cases, the prognosis depends chiefly on the extent of the paralysis and the electrical irritability of the paralyzed muscles. Cases which approach generalized lead paralysis offer a more serious prognosis.   In muscles which have lost their faradic excitability, recovery cannot be expected before the end of three or four months, if at all.

6. Prophylaxis is an important feature in treatment, and chiefly involves the proper construction of factories.   After the paralysis has developed, potassium iodide (gr. xij. t. i. d.) may be given internally. Annuschat has recently shown that this facilitates the excretion of lead from the body.   In addition, warm baths, particularly sulphur baths (30° R., fifteen to thirty minutes' duration).   We may also apply the

galvanic spinal current (large electrodes, vigorous current, at first the anode for two to three minutes, then the cathode to the cervical enlargement).   Also the galvanic current labile, the anode being placed in the cervical enlargement, the cathode stroked methodically over the individual muscles.   The faradic current may be employed if the muscles retain their faradic excitability.   Galvanization of the sympathetic has also been recommended.

b. Arsenic Paralysis.

1. Arsenic paralysis is more often the result of acute than of chronic poisoning, and may develop in a few days.   It is distinguished from lead paralysis by affecting chiefly the lower limbs, by the more rapid atrophy of the paralyzed muscles, and the prominence of sensory disturbances.

2. As a rule, there is paraplegia of the lower limbs, more rarely of all the limbs or the upper limbs alone.   The paralysis and atrophy of the muscles are often preceded by violent pains; and numbness, formication, and analgesia are noticed not infrequently.   The electrical irritability, the cutaneous and tendon reflexes present the same changes as in lead paralysis.   In one case, Seeligmueller observed marked tabetic symptoms.

3. In pieces of the muscle, which had been removed from the patient by harpooning, Da Costa noticed narrowness of the muscular fibres, increase of the nuclei of the sarcolemma, in other parts waxy degeneration, slight fatty degeneration, increase of the interstitial connective tissue, and accumulation of fat in it.   Nevertheless, Da Costa assumes a spinal origin of the paralysis.   In their experimental investigations, Mierzejewski and Popof observed poliomyelitis, but the process often extended into the white matter.

4. The treatment is the same as in lead paralysis.

c. Phosphorus Poisoning.

Acute and chronic phosphorus poisoning may also give rise to paralyses, in the form of partial paralysis, monoplegia, and paraplegia.   Pains, paræsthesiæ, and objective sensory disturbances have also been observed in the paralyzed parts.   The situation of the lesion is not known with certainty.   Degeneration of the peripheral nerves has been observed, and likewise changes in the spinal cord.   Danillo and Vulpian have recently made experiments on dogs.   In acute poisoning, they found hyperæmia and an accumulation of pigment in the vessels of the anterior horns, and swelling and the development of vacuolæ in the ganglion cells themselves; in chronic poisoning, the white matter also took part in the process.

d. Carbonic Oxide Gas and Bisulphide of Carbon Paralysis.

Paralyses have been described after a number of cases of carbonic oxide poisoning.   These paralyses may be peripheral, spinal or cerebral in origin, and are probably the result of hemorrhages with secondary inflammatory and degenerative changes.

These remarks hold good concerning paralysis after poisoning with bisulphide of carbon, to which workmen in India-rubber factories are especially liable.

e. Paralysis after Ergot Poisoning.   Ergotism.
(Raphania.   Morbus Cerealis.)

Paralyses after ergot poisoning are almost always spinal in character.   In three cases (in one family) under my observation, marked atrophy and contractures of the muscles had developed.   Little is known concerning the site of the disease.   Tuczek recently described very marked changes in the posterior columns; the knee jerk was absent in these cases during life, and the Brach-Romberg symptom and ataxia had also been observed in some cases.   Treatment according to general principles.

Pellagra and ergotism are probably identical.

f. Alcohol Paralysis.

Spinal paralytic symptoms may develop in acute, more frequently in chronic, alcoholism. It is well known that some individuals, when intoxicated, keep a clear head, while the feet become "groggy." Certain alcoholic drinks have a special power of affecting the legs.

Paraplegia, ataxia, and cutaneous hyperæsthesia have been observed in chronic alcoholism. The latter are perhaps associated with meningitic changes. The condition of the tendon reflexes is indicated on page 111. The symptomatology may be very similar to that of tabes (pseudo-tabes). The treatment consists in gradually weaning the patients from their bad habits.

8. Writer's Spasm. Mogigraphia.

(Graphospasm. Cheirospasm.)

I. ETIOLOGY.—Writer's spasm is most frequent from the twentieth to fortieth years; it never occurs in childhood, and is rare in old age. The disease almost always attacks men.

The causes are variable. In some patients there is a congenital nervous disposition, inasmuch as epilepsy, hysteria, and other neuroses are observed among the relatives, and perhaps the patient himself was nervous since youth.

In other cases acquired nervousness constitutes the predisposition to writer's spasm, for example, in masturbators and those who indulge in excesses in Baccho et Venere.

Strong mental excitement may produce or intensify the disease.

Some patients attribute their complaints to cold or injury, for example, a wound of the finger, running a needle into it, wearing tight gloves, etc. ·

In rare cases an inflammation of some of the nerves of the brachial plexus has acted as the cause of the disease. Runge describes a case which resulted from periostitis of the arm.

The most frequent cause, however, is strain or a bad position of the hand in writing.

It is stated not infrequently by the patients that the first symptoms appeared while making the balance sheet at the end of the year. The disease has also been attributed to the use of steel pens, but this cannot be the sole cause since it was observed before their introduction.

II. SYMPTOMS.—As a rule, the symptoms develop very gradually. There are sometimes certain prodomata, such as a feeling of pressure in the head, depression, excitement, poor sleep, nervous dyspepsia, etc. The manifest symptoms begin as more or less impediment to the act of writing, but after a time (sometimes not until the lapse of months and years) writing becomes impossible.

The first muscles affected are those employed in writing, viz., the interossei and lumbricales, the muscles of the ball of the thumb, and the extensors and flexors of the forearm. The spasm may also extend to the muscles of the arm, shoulder, and even the neck. Sometimes only a few, in other cases many groups of muscles are affected.

Benedikt distinguishes spastic, tremulous, and paralytic writer's spasm.

The spastic form is the most frequent, the paralytic variety the rarest. One form is sometimes complicated by another.

Spastic writer's spasm consists of tonic, more rarely clonic muscular spasms, which impair or prevent the sure manipulation of the pen.

In some cases there is flexion spasm of the thumb, so that, on attempting to write, the thumb is drawn spasmodically into the palm of the hand. In other cases there is spasmodic flexion or extension of the index finger. If the extensors of the thumb and index finger are affected at the same time, the pen will drop from the hand. The spasm sometimes affects the extensors or the abductor and opponens digiti minimi, and is associated with such distressing pain that it becomes impossible to continue writing. Spasm of the flexors of the wrist joint is not infrequent. If it occurs in the flexor and extensor carpi ulnaris, the hand is drawn forcibly across the paper. Various other combinations of spasm may also be observed.

Tremulous writer's spasm is characterized by trembling movements which occur only during writing.

Paralytic writer's spasm is really writer's paralysis. It is shown by a feeling of increasing exhaustion and tension which develops during the act of writing, and becomes so severe that the hand is held quiet on the paper.

The disturbances of co-ordination in question sometimes occur at each attempt to write, in other cases only after prolonged writing. Mental excitement exercises a marked influence upon the disease.

The gross power of the muscles affected by spasm is in no wise impaired. Many patients can also perform, without difficulty, other delicate manipulations, such as threading a needle, sewing, playing piano, etc.

In addition to writer's spasm, the patient may also suffer from spasm of other groups of muscles; viz., strabismus, stuttering, pharyngeal spasm, spasm in the muscles of the back of the neck, also tremor and paresis of the legs.

The affected muscles are sometimes tender on pressure.

Complaint is made not infrequently of paræsthesiæ: a feeling of coldness, burning, formication, etc. Some authors have noticed vaso-motor disturbances (pallor of the fingers and a feeling as if they were dead) at the beginning of the spasm. The patients sometimes complain of neuralgiform pains, most frequently in the distribution of the ulnar nerve, next in that of the median nerve. The nerves of the arm are occasionally tender on pressure, and neuritic swellings have sometimes been observed upon them.

Painful pressure points are sometimes found on certain parts of the bones of the arm or on some of the cervical spinal processes.

The electrical irritability of the affected muscles is generally unchanged; it is sometimes slightly diminished, more rarely increased. Gowers describes abolition of electro-muscular and electro-cutaneous sensibility. Eulenburg has observed qualitative changes in the electrical irritability of the nerves supplying the affected muscles (predominance of AnClC and early appearance of AnOC, and CaOC).

The disease continues to grow worse unless the patients are able to give up writing for a long time.

The more the disease progesses, the more the written characters are changed. The writing often looks as if written by a beginner, or while riding over a rough road, and finally legible characters can no longer be made.

Many patients help themselves at first by using thick penholders or quills, by writing with the arm elevated or with changed position of the

wrist-joint and fingers. Others learn to write with the left hand. But, unfortunately, the same symptoms develop not infrequently in the left arm, or writing with the left hand causes very annoying spasmodic conditions in the right arm.

III. PATHOGENESIS.—The disease is probably the result of purely functional disturbances. These are evidently situated in the spinal cord, because the act of writing requires the delicate co-ordination of adjacent groups of muscles, and the co-ordinating centres of these muscles are situated in the cervical enlargement of the cord. Abnormal irritability, ready exhaustion, and irregular stimulation of these centres suffice to explain the symptoms of writer's spasm. In some cases, however, the primary disturbance is situated in the brain, as is shown by the feeling of pressure in the head, vertigo, mental depression, etc. In other cases the starting-point is at the periphery (neuritis, periostitis, etc.).

IV. DIAGNOSIS.—The diagnosis is easily made. We must always endeavor to determine the variety of the disease, and for this purpose must carefully study the disturbances during the act of writing. We should also search carefully for pressure points.

V. PROGNOSIS.—The prognosis is not very favorable. Recovery is rare unless the patient abstains permanently from writing.

VI. TREATMENT.—Writing must be strictly prohibited for many months. If hereditary factors or an acquired nervous disposition enter into the etiology, we should prescribe potassium bromide, iron, quinine, cold-water cures, a stay in the country, and careful massage of the muscles. Injurious habits must be given up, and peripheral lesions (injury, periostitis, neuritis) treated in the ordinary way. If pressure points are present on the spinal column, the anode of the galvanic current should be applied to them (cathode to the sternum, daily sitting for five minutes, current not too strong).

Treatment with the galvanic current likewise offers the best hopes of success under other circumstances. If cerebral symptoms are prominent, feeble currents should be passed transversely and obliquely through the skull. A spinal current may also be applied to the cervical cord. Peripheral galvanization may be applied to the affected muscles and nerves—the labile current in the tremulous and paralytic varieties, the stabile current in the tonic form (according to Eulenburg the anode is preferable). Galvanization of the cervical sympathetic has also been recommended. M. Meyer obtained good results from faradization of the individual muscles, but this must be avoided in the spastic variety. Erb applied permanently a simple galvanic element (copper and zinc plates connected by a wire and placed over a nerve trunk of the arm upon a moist piece of linen). The treatment must be continued for a long time in all cases.

Good results have been obtained recently from massage of the affected muscles and gymnastics, but it is advisable to have the treatment carried out by a skilled specialist.

Numerous other measures have been employed in this disease: a, alcoholic and narcotic inunctions, douches, blisters, cups, derivatives of all kinds, ice-bag, ether irrigation of the spine; b, narcotics and nervines; c, mechanical apparatus (thick, cork penholders, rings to fasten the penholders, bandages to the wrist-joint); d, nerve stretching.

APPENDIX.—Similar disturbances may occur in other fine manipulations which necessitate the co-ordinated action of groups of muscles. The gross power of the muscles is unchanged, but their power of co-ordinated action is lost (co-ordinating professional neuroses).

Most closely allied to writer's spasm is telegrapher's spasm. Pianist's spasm occurs particularly in young ladies, and usually in the right hand. Violinist's spasm may affect either hand. Tailor's and shoemaker's spasm is manifested in the muscles of the hand and arm, and renders impossible the use of the scissors, needle, etc. Similar conditions are observed in smiths, masons, sawyers, weavers, watchmakers, printers, etc., and also in the lower limbs, in turners, sewing machine operators, dancers, etc.

Spasm of the larynx has been described in players on the clarionet.

9. *Tetany.*

I. ETIOLOGY.—Tetany manifests itself by the occurrence of tonic muscular spasms, which come and go in paroxysms, affect certain groups of muscles, and are associated with increased electrical and mechanical excitability of the corresponding motor nerves.

The disease occurs most frequently at a youthful age. It is not infrequent in teething children, later it occurs particularly at the period of puberty (thirteenth to thirtieth years). It is rare in later life.

Rachitic, pale, and feeble individuals exhibit a predisposition to the disease.

Some authors attribute a certain degree of influence to heredity. Murdoch observed tetany in several brothers and sisters, and Bouchut states that it is particularly frequent in children whose parents are nervous and suffer from neuroses.

Among the not infrequent direct causes are colds, particularly from sleeping in damp rooms, camping out, etc. The influence of rheumatic causes also appears to be shown by the fact that tetany is often associated with swelling of the joints, and that it is especially frequent in the cool months.

It often appears to be the reflex effect of peripheral irritation (teething, worms, overloading of the stomach and intestines, suppression of the menses, pregnancy, lactation, operation for goitre).

Tetany sometimes follows acute infectious diseases or exhausting diseases. These conditions probably produce, in many cases, merely a predisposition, and other influences are necessary to produce the disease itself.

It is sometimes the result of violent excitement. A number of cases have been reported in which tetany occured as an epidemic in schools and similar institutions.

II. SYMPTOMS.—The attacks of spasm are almost always preceded by certain prodromata: dragging and tearing pains in the limbs, a feeling of coldness, formication, etc. The prodromata may last for days and weeks. In rare cases cerebral symptoms occur: vertigo, ringing in the ears, a feeling of confusion.

The tonic muscular spasms sometimes follow the prodromata spontaneously, and in certain patients are apt to occur at night, or they appear after bodily strain or mental excitement.

In typical cases they are confined to the flexors of the fingers and wrists; they are almost always bilateral, exceptionally unilateral. They often extend to the flexors of the forearm and the adductors of the arm. In exceptional cases the spasm affects the extensors of the fingers and wrist, or both the flexors and extensors.

The muscles of the lower limbs are sometimes affected, sometimes only the plantar flexors of the toes, or the calf muscles, occasionally even the extensors and adductors of the thigh.

The spasm extends more rarely to the muscles of the back, abdomen, and chest, to the muscles of the pelvis and diaphragm. Indeed, the spasm finally may become general, and affect the muscles of the face, larynx, pharynx, tongue, œsophagus, and detrusor of the bladder.

In typical cases, the position of the affected fingers and hands is so characteristic that it almost suffices to warrant a diagnosis. Trousseau compares it to the position assumed by the obstetrician when he attempts to introduce the hand into the vagina.

The thumb is strongly adducted and bent into the palm of the hand; the other fingers, particularly the fourth and fifth, are strongly flexed in the metacarpo-phalangeal joint, and extended in the phalangeal joints; the palm of the hand is deeply hollowed, and the entire hand is strongly flexed towards the ulnar side. If the flexors of the forearm are affected, the forearm appears semi-flexed; in spasm of the adductors of the arm, the arms may be so strongly drawn against the thorax that the flexed forearms cross each other over the epigastrium.

In spasm of the flexors of the toes, the great toe is drawn under the adjacent toes; spasm of the calf muscles is shown by pes equinus, pes varo-equinus, more rarely by pes valgo-equinus. In the knee-joints, the lower limbs are usually extended, and the thighs are adducted against one another. In tonic spasm of the muscles of the back, the spinal column appears strongly curved anteriorly. Spasm of the thoracic muscles and diaphragm may give rise to threatening symptoms of suffocation. Spasm of the muscles of the neck and throat causes cyanosis of the face and protrusion of the eyeballs as the result of venous stasis.

The contours of the muscles often stand out sharply beneath the skin. Fibrillary contractions are sometimes visible during the spasm. The muscles are firm, and more or less tender on pressure.

The contractures can usually be overcome after a certain degree of effort, but the limb returns to its former position as soon as it is released.

The spasmodic attacks vary in duration from a minute to one or more hours, and even three days. The contractures persist, though less vigorously, during sleep.

The number of attacks also varies greatly. Sometimes single attacks occur at intervals of days, weeks, and months, and occasionally the disease terminates with one paroxysm. In other cases, numerous seizures recur in the course of a single day.

During the attack, the majority of patients suffer from annoying sensations: a feeling of coldness, prickling, formication, etc. Some complain of a painful feeling of tension in the contracted muscles, others of neuralgiform pains, which may be confined to certain nerve tracts; central symptoms may also be noticed (vertigo, pressure in the head, ringing in the ears, etc.).

Great diagnostic significance attaches to three other symptoms, viz., Trousseau's phenomenon, the increase of the electrical and of the mechanical irritability of the motor nerves.

Trousseau's phenomenon is the ability to produce a seizure of tetany by pressure upon the nerve trunks or arteries of the arm. The attack occurs after the pressure has been exercised one to two minutes, and disappears when the pressure is discontinued. In some cases, pressure on the artery alone produces an attack, while compression of the nerve is useless. This symptom is very rarely absent, and so long as it can be produced, we must be prepared for the recurrence of spontaneous attacks of tetany.

A few cases have been reported in which pressure on other parts of the body also produced attacks, for example, compression of the cervical sympathetic and

carotid, pressure and faradic irritation of painful points on the spine, or pressure on the muscles of the wrist joint and on the radial artery. Czerny observed an attack follow elevation of the arm. The symptom is produced with more difficulty in the lower limbs by pressure on the crural artery or sciatic nerve.

The electrical irritability of the motor nerves is increased. A faradic current of very slight strength, when applied to the nerve, suffices to produce muscular contractions. On the application of the galvanic current (to the nerve) CaClC and AnOC occur very early, CaClTe and AnClTe soon follow, and it is a peculiar fact that AnOTe is produced without difficulty. Chvostek claims to have observed CaOTe in two cases. These remarks hold good concerning the nerves alone, not concerning the muscles supplied by them. The facial nerve, as a rule, remains unaffected.

So long as these electrical changes persist, the attacks of tetany may relapse.

The mechanical irritability of the motor nerves is increased *pari passu* with the electrical irritability, so that gentle tapping on the nerve will produce contractions in the corresponding muscles. As a rule, the facial nerve is not affected.

The appearance of the parts affected with tetany is often unaltered, in some cases swelling of the joints with redness and œdema has been observed.

The cutaneous sensibility is generally changed, but sometimes very slightly. Manouvriez observed partial anæsthesia even in the intervals between the attacks.

Hasse states that the more marked the anæsthesia the more severe the attacks of spasm will be. He also observed abolition of the muscular sense, so that the patients could only hold objects by the aid of the sense of sight. Painful pressure points on the spine are reported in a number of cases.

The general condition is often unaffected. Some patients complain of dyspepsia and drowsiness, or, on the other hand, of excitement. The height of the attack is sometimes accompanied by profuse perspiration or fever, or both.

As a rule, the disease terminates in recovery, though slight contractures and pareses may persist for a long time. Kussmaul observed retinitis as a sequel. In children, the height of the disease may be attended with fatal eclampsia. Death may also result from increasing exhaustion and persistent diarrhœa.

III. ANATOMICAL CHANGES AND PATHOGENESIS.—Nothing is known concerning the anatomical changes in tetany. Hemorrhages into the spinal meninges, increase and sanguinolent color of the spinal fluid, softening and sclerosis of the cord, minute hemorrhages into the cord, and thickening of the vessels, have been observed in a number of cases; but these are either accidental complications, or the secondary effects of the tetany.

It is now generally conceded that the disease is neuropathic in its origin, but it is questionable whether the starting-point is in the nerves, spinal cord, brain, or sympathetic. The cerebral theory is opposed by the fact that, in the majority of cases, cerebral symptoms are absent, and the cerebral nerves are usually unaffected. The increased electrical and mechanical excitability of the motor nerves seems to point to its peripheral origin, but this may also be a secondary effect of spinal changes. If we take into consideration that the disease is often produced in a reflex manner (necessarily through the medium of the spinal cord), and that pressure points are found on the spine, it seems plausible to assume a spinal origin of the disease, and to explain it by increased irritability of the ganglion cells of the anterior horns, upon which depend the secondary changes in the peripheral nerves. N. Weiss thinks that the sympathetic is the starting-point of the disease, and that its irritation gives rise to the circulatory changes in the cord.

IV. DIAGNOSIS.—The diagnosis is easy if we bear in mind the cardi-

nal symptoms: intermittent tonic spasms in certain groups of muscles, Trousseau's phenomenon, and increased electrical and mechanical excitability of the nerves.

The disease is distinguished from tetanus by the fact that the latter first manifests itself by trismus. In hysterical spasms and the co-ordinating professional neuroses, the mechanical and electrical irritability of the nerves is not increased. Some cases of spasmodic ergotism possess very great similarity to tetany, but are distinguished from the latter by the etiology.

V. PROGNOSIS.—The disease almost always terminates in recovery, although months may elapse before it disappears entirely. The patient cannot be regarded as completely cured until Trousseau's phenomenon and the increased excitability of the motor nerves have disappeared. Trousseau recognized three degrees of severity of the disease: in the mildest, it is confined to the muscles of the limbs and general symptoms are absent; in the second grade, the muscles of the trunk are affected, the attacks are more frequent and violent; in the third, there are disturbances of the general condition.

VI. TREATMENT.—Our attention must first be directed to the removal of the cause. In rheumatic cases, we order potassium iodide, salicylic acid, and sweat baths; in suppressio mensium, warm foot baths and leeches to the cervix uteri, etc.

Narcotics and nervines have been recommended against the disease itself, but no single remedy is especially efficacious.

We may mention: potassium bromide, chloral hydrate, opium, belladonna, curare, chloroform, ether, valerian, arsenic, etc. The remedies may be administered in various ways.

Cups, blisters, ice-bags to the spine, lukewarm baths, cold-water cures, etc., have also been recommended.

Good results have been obtained from the use of electricity, but no single method of application seems to be especially useful.

If there are painful spinal points, the anode of a constant galvanic current (not too strong) may be applied to them, the cathode to the sternum; each sitting two to four minutes, every other day. The anode may also be applied to the cervical or the lumbar enlargement. Or the peripheral nerves are galvanized labile with the anode, which is slowly stroked towards the nerve plexus. Anodal applications may also prove effective during the spasmodic seizures. If the faradic current is employed at all, it should be applied as a feeble current to the spine and nerve trunks.

10. *Saltatory Spasms.*

Seven cases of this disease have been reported. It occurred particularly in nervous and hysterical individuals with a neuropathic hereditary taint. Upon bringing the foot to the ground, and in standing and walking, clonic muscular contractions occur of such a vigorous character that the entire body undergoes a hopping, dancing movement. The spasms were confined to the lower limbs, or they also involved the upper limbs and the muscles of the trunk and back of the neck. They were accompanied by drawing pains, and in two cases occurred in paroxysms and were accompanied by a sort of aura. No paralysis, anæsthesia, or other severe spinal symptoms. Some patients complained of spinal tenderness, and Guttmann could produce attacks by pressure on the spine. There is evidently increased reflex excitability of the ganglion cells of the anterior horns in this affection. Frey attributes it to a definitely localized myelitis; Erb is inclined to regard some of the cases as spastic spinal paralysis. Spontaneous

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improvement occurred in some cases, while others were incurable. Warm baths, ice and wet cups to the spine, narcotics, nervines, and electricity have been employed in treatment.

11. *Primary Spinal Muscular Spasms.*

(*Thomsen's Disease.*)

I. ETIOLOGY.—In this disease, tonic spasm of the muscles occurs during voluntary movements, and either delay or entirely abolish the execution of the intended movement. The disease is also known as myotonia congenita, tonic spasms in voluntarily moved muscles, and hypertrophic spastic spinal paralysis.

Heredity plays a very important part in the etiology. In Thomsen's family the disease occurred in five generations. Schoenfeld observed the development of the disease as the result of fright. Other causes are unknown.

II. SYMPTOMS.—The symptoms are confined chiefly to the voluntary muscles, but Seeligmueller observed in one case formication and a feeling of coldness in the limbs, in another case absence of the tendon reflexes. The bladder and rectum were always unaffected.

The muscles of the lower limbs were most affected, next those of the upper limbs. In some cases the muscles of the tongue, face and eyes, and of mastication were also involved.

In this disease, voluntary movements cannot be carried out immediately because tonic spasm develops in the muscles which are to be moved. If the patients wish to rise and walk, they feel a disagreeable feeling of tension; some time elapses before the resistance is overcome. The spasms may be so violent that the patients fall and roll involuntarily about the floor. If they have grasped an object with the fingers, they are unable to let it drop at once. Affection of the tongue is shown by hesitating speech and awkwardness in mastication.

Resistance is also experienced during passive movements.

The muscles are often unusually large, generally feel very firm, and, on mechanical and faradic irritation, form thick ridges which persist for a long time. The faradic and galvanic excitability is unchanged. Fibrillary contractions are sometimes observed in the muscles.

Continued movement and warmth diminish the spasms, mental excitement and cold intensify them.

The first symptoms are often noticeable in the cradle. During childhood the patients attract attention by their awkward manner; in rare cases the symptoms do not begin until the twentieth year or later.

The disease continues for life, but remissions and exacerbations have been described.

III. ANATOMICAL CHANGES.—In one case, examination of a piece of excised muscle showed nothing beyond somewhat unusual width of the fibrillæ. The disease is generally regarded as spinal in its origin, but nothing further is known of its nature.

IV. DIAGNOSIS.—The diagnosis is easy. It is distinguished from muscular hypertrophy by the absence of changes of electrical excitability and the presence of spasms.

V. TREATMENT is of no avail. Some relief is said to have been obtained by gymnastic exercises. Trial should be made of baths and electricity.

C. DISEASES OF THE SPINAL MENINGES.

1. *Inflammation of the External Surface of the Dura Mater.* *External Spinal Pachymeningitis.*

(*Peripachymeningitis. Perimeningitis Spinalis.*)

I. ANATOMICAL CHANGES.—The inflammatory process in question is situated on the outer surface of the dura mater in the loose, fatty connective tissue which separates the dura mater from the inner surface of the spinal canal. In the majority of cases the inflammation is circumscribed, hardly occupying more

than the height of one to three vertebræ; more extensive inflammations are rare. In some cases there are several separate foci of inflammation.

The inflammation is most marked on the posterior surface, chiefly because the connective tissue is most abundant in that locality, perhaps in part on account of the influence of the force of gravity. It rarely extends beyond the cervical region, because the extradural cellular tissue gradually diminishes in the upper part of the cord.

In a series of cases the inflammatory products constitute a thickening and cellular inflammation on the outer surface of the dura mater and in the peridural cellular tissue. This may be associated with more or less swelling and redness at

Fig. 82.
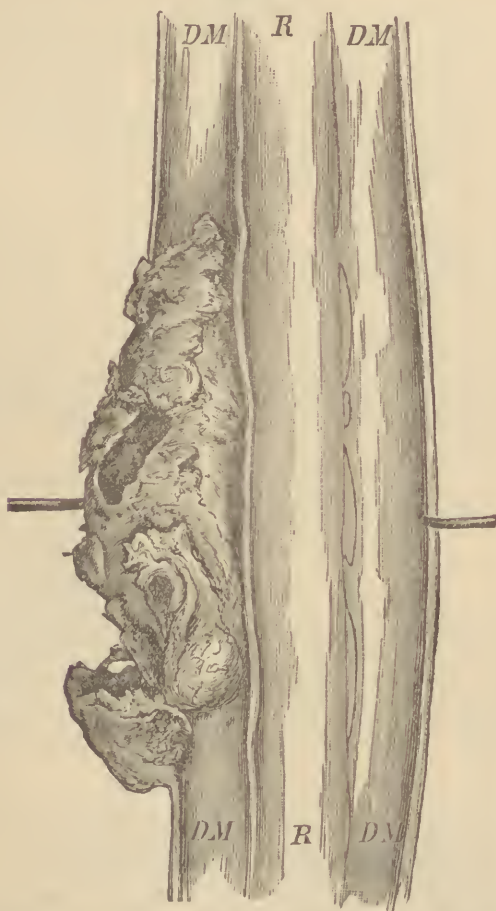
External spinal pachymeningitis at the level of the fifth to seventh dorsal vertebræ. Natural size. Spinal cord seen from behind. Dura mater (*DM*) drawn to one side, on its outer surface a cheesy mass on the left side. Symptoms of compression of the cord (*R*) during life. Starting-point of the lesion in a right-sided cheesy pleurisy, which extended through the intervertebral foramina to the spinal canal.

the site of disease. In other cases the outer surface of the dura mater is covered with a thick, callus-like membrane, which is almost fibrinous in appearance. Well-marked abscesses are found in some cases. Dry, cheesy, tuberculous masses of considerable size may also be produced (vide Fig. 82). In chronic cases fibrous thickenings and adhesions to the outer surface of the dura mater are found.

The inflammation extends not infrequently to the inner surface of the dura

mater, giving rise to swelling, redness, infiltration, abscess, or cheesy tuberculous processes. Even the arachnoid and pia mater may take part in the inflammation.

In a series of cases the process results in simple compression of the cord and nerve roots, in others it gives rise to compression myelitis (vide page 145).

II. ETIOLOGY.—External spinal pachymeningitis is almost always a secondary condition, indeed it is questionable whether it is ever primary. The primary morbid process is generally situated near the spine, whence the inflammation extends through the intervertebral foramina to the peridural space. It has been observed after tuberculosis or inflammation of the vertebræ, pleuritis, peripleuritis, syphilitic ulcers of the pharynx, suppuration of the cellular tissue of the neck, psoas abscess, and deep spreading decubitus. It may also be produced by other conditions, particularly suppurations in the thorax, peritoneum and pelvis, and in migrating neuritis. Syphilis must also be mentioned as a cause.

III. SYMPTOMS.—The symptoms are very similar to those of spinal meningitis.

Most patients complain of stiffness in the back, which is especially noticeable while sitting or standing, or on rotating the spine.

The spine is tender on pressure over parts corresponding to the inflammation. A blow on the head or shoulders, the passage of a sponge dipped in hot water, or the slow labile application of the cathode of a galvanic current causes pain exclusively or chiefly in these localities.

Visible or palpable changes in the spine (swelling, redness, fluctuating tumors, etc.) are noticeable if the affection is secondary to disease of the spinal column.

The patients often complain of a cincture feeling around the trunk, corresponding to the approximate site of disease.

Violent pains—usually in paroxysms—are experienced. either along the spine or radiating into the extremities. Hyperæsthesia, hyperalgesia, and paræsthesiæ are often observed. In addition, there may be tonic or clonic twitchings and contractures in the muscles of the extremities. Paralysis and anæsthesia may be looked for, if pressure on the nerve roots has interfered with conduction. The reflex irritability is then destroyed, and the paralyzed muscles undergo atrophy. The electrical irritability of the paralyzed muscles is the same as in peripheral paralysis (vide page 7).

If the cord is compressed, motor and sensory paraplegia will develop below the site of compression, with paræsthesia, increased reflex excitability, vesical disturbances (at first retention, later incontinence), perhaps incontinence of fæces and decubitus (vide page 146). The disease generally runs a subacute or chronic course. Fever is occasionally noticeable, often as the result of the primary disease itself.

IV. DIAGNOSIS.—The diagnosis is not easy. The chief points to be considered are the etiology and the irritative symptoms mentioned above. It is distinguished from spinal meningitis by the absence of rigidity of the neck, since the cervical region is almost always unaffected.

V. PROGNOSIS.—The prognosis is always grave. The majority of cases terminate fatally, but Leyden has shown that recovery is possible.

VI. TREATMENT.—Treatment must first be directed towards the removal of the causes. In other respects it is the same as that of spinal meningitis (vide page 170).

2. Inflammation of the Inner Surface of the Spinal Dura Mater. Internal Spinal Pachymeningitis.

Internal spinal pachymeningitis generally runs a chronic course, and occurs as the hypertrophic or hemorrhagic varieties.

a. Hypertrophic internal spinal pachymeningitis consists of inflammatory proliferations and fibrous thickenings on the inner surface of the dura mater. In the majority of cases similar changes have developed upon the arachnoid and pia mater, so that the spinal cord is constricted by a ring of connective tissue which sometimes attains a thickness of one cm., often presents concentric lamellæ, and contains numerous interstitial spaces (vide Fig. 83). The proliferation is generally most marked posteriorly, where there are usually extradural adhesions to the posterior ligament. The changes are generally circumscribed, the lower half of the cervical enlargement being most frequently affected (pachymeningitis cervicalis hypertrophica).

The dangers of the disease consist in the constriction of the nerve roots and

spinal cord. The symptoms of compression of the cord are gradually followed by those of compression-myelitis. The gray matter will be affected with special severity, and it is perforated by new-formed canals (vide Fig. 83). The latter are found chiefly in the gray commissure, are lined with a membrane, and contain serous fluid.

FIG. 83.

Transverse section through the middle of the cervical enlargement in hypertrophic, internal spinal pachymeningitis. After Charcot. *A*, thickened dura ; *C*, new-formed cavities in the gray matter ; *B*, nerve roots.

The disease is rare. It has been attributed to colds, and living in damp places.

Symptoms will not be produced unless the nerve roots are compressed and later paralyzed, or the cord itself undergoes compression. Charcot distinguishes a stage of irritation (lasting two to three months), and a stage of paralysis and atrophy.

FIG. 84.
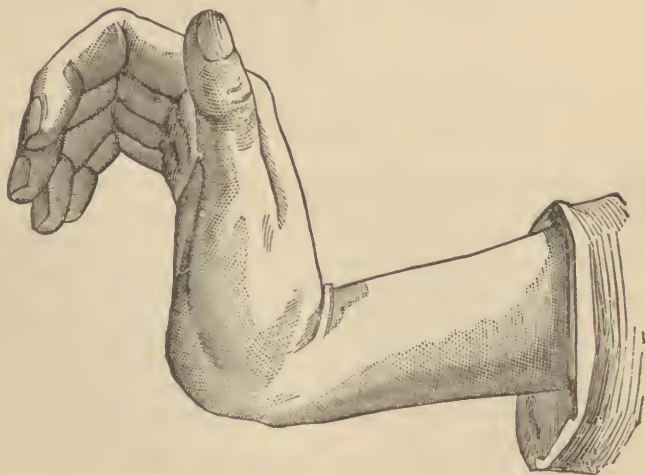
Most frequent position of the hand in hypertrophic cervical pachymeningitis. After Charcot.

In the stage of irritation, the patients complain of pains in the upper part of the spine, the occiput, between the shoulders, and in the arms (particularly in the large joints). Pressure on the spine does not give rise to pain. The majority of the patients complain of stiffness in the back of the neck, and a feeling of constriction in the upper part of the chest. The pains appear in paroxysms or in-

crease periodically in intensity. Hyperæsthesia, and occasionally paræsthesiæ, are noticeable in the upper limbs. Vesicular eruptions, marked desquamation and roughness of the epidermis may also make their appearance. The muscles not infrequently present tonic or clonic contractions, rigidity, and contractures. These symptoms are evidently the result of irritation of the nerve roots which pass through the hyperplastic portions of the dura mater.

The second stage is characterized by paralysis of muscles in the upper limbs, fibrillary contractions and atrophy of the paralyzed muscles, changes in electrical irritability (such as occur in peripheral paralysis), and anæsthesia. As a rule, the ulnar and median nerves are paralyzed, the intact extensors of the forearm acquire the predominance and produce permanent dorsal flexion of the hand, while the fingers are flexed at the second and third joints (vide Fig. 84). If the morbid process is situated very high, the radial nerve will be paralyzed, and the hand will permanently assume a flexed position (vide Fig. 85). All these symptoms are the result of compression and complete interruption to conduction in the spinal roots.

If the spinal cord itself becomes compressed, paretic and paralytic symptoms will develop in the lower limbs. The muscles undergo atrophy merely as the result of disuse, and their electrical irritability is unchanged. We will find increased reflex excitability, diminished sensibility, disturbances of the bladder, decubitus, etc.

FIG. 85.

Position of the hand in radial paralysis, as the result of hypertrophic cervical pachymeningitis.
After Ross.

The disease often lasts for many years, but recovery is possible. The complication with pulmonary phthisis is relatively frequent.

The diagnosis is not very difficult. The second stage may be mistaken for progressive muscular atrophy, but the latter is not preceded by an irritative stage, begins generally in the interossei, the thenar and hypothenar eminences, and sometimes extends to the nerve-nuclei in the medulla, and gives rise to bulbar paralysis; hypertrophic pachymeningitis hardly ever extends to the medulla oblongata, and although it causes paralysis of the lower limbs, the muscles do not undergo rapid atrophy or changes in electrical excitability.

In amyotrophic lateral sclerosis there is no stage of irritation, sensory disturbances are absent, the disease runs a more rapid course, affects the upper and lower limbs in a uniform manner, and almost always proves fatal by extension to the medulla oblongata.

The differential diagnosis must also be made from tuberculosis of the spine, tumors of the spine and meninges, and external spinal pachymeningitis; the chief points to be considered are pain and deformity in the spine, tuberculosis of the lymphatic glands and lungs, and tumors in other organs.

The treatment is similar to that of spinal meningitis.

b. *Hemorrhagic internal pachymeningitis* corresponds exactly to hæmatoma of the cerebral dura mater. The inner surface of the dura mater is covered with more or less extensive, extremely vascular membranes. These may often be separated into several layers, between which is situated partly fluid, partly clotted blood. Under the microscope we find hæmatoidin crystals and red blood-

globules in various stages of destruction. The extravasations may be so large as to compress the nerve-roots and spinal cord. The pia mater sometimes takes part in the hemorrhage, and the spinal fluid is tinged with blood. In some cases the change extends along the entire length of the cord, in others it is confined to small, scattered foci.

Similar changes are generally found on the cerebral dura mater, because both conditions are the results of the same causes. They are observed in the insane and in drunkards; Leyden reports one case after injury.

In many cases the disease remains latent, in others it produces the symptoms of irritation or paralysis of the nerve-roots or spinal cord. These symptoms may be attributed to hemorrhagic pachymeningitis if the etiological factors are present. Sudden signs of meningeal hemorrhage may develop if vessels in the new membranes rupture and produce an extensive hemorrhage.

The treatment is similar to that of spinal meningitis.

3. *Acute Spinal Meningitis.*

I. ETIOLOGY.—Inflammations of the arachnoid (arachnitis) and pia mater (piitis) are almost always associated with one another, and are known as spinal meningitis or leptomeningitis. They are sometimes confined to the cord or to parts of it, sometimes they extend to the cerebral meninges or vice versa (cerebro-spinal meningitis). We will here discuss spinal meningitis alone.

Colds (sleeping on the damp ground, working in water, etc.) and injuries are mentioned not infrequently as causes. Spinal meningitis is sometimes the result of simple concussion or lifting heavy weights, in other cases it is produced by fracture or dislocation of the vertebræ, stab or gunshot wounds, etc.

Secondary inflammations are more frequent. In Cruveilhier's case a pulmonary cavity perforated the spinal canal and set up spinal meningitis. It may also be the result of any of the etiological factors which give rise to external spinal pachymeningitis (vide page 164).

Spinal meningitis occurs occasionally in connection with infectious diseases: pneumonia, typhoid fever, cholera, dysentery, etc. Hasse observed several cases after articular and muscular rheumatism.

It is more frequent in the male sex, particularly in childhood and middle age.

II. ANATOMICAL CHANGES.—These are divided into the stage of hyperæmia and beginning exudation, that of the completed exudation, and that of resolution.

In exceptional cases death occurs in the first stage. The pia mater and arachnoid tissue are unusually red, swollen, and succulent. The redness is diffuse and uniform, or depends upon unusually distended, finer blood-vessels. Here and there are more or less numerous and extensive extravasations. The larger vessels are sinuous and distended to the point of bursting. The surface of the pia mater and arachnoid tissues looks loosened and velvety. The spinal fluid is cloudy and contains flocculi.

The second stage begins with the deposit of purulent or fibro-purulent flakes. These continue to increase, and finally cover the surface of the pia mater, the arachnoid tissue, and even the inner surface of the dura mater with purulent or fibrinous deposits, which are often dotted with blood. These changes are circumscribed or diffuse, according to the etiological factors. They are most marked on the posterior surface (influence of the force of gravity), and diminish towards the medulla ob-

longata.   Even if cerebral meningitis is also present, the medulla often
escapes entirely, or almost entirely.

In the third stage, the purulent masses are gradually absorbed, but
thickenings and abnormal adhesions of the meninges usually remain as
permanent residua.

As a rule, the inflammation is not confined to the pia mater and arach-
noid tissue, but attacks important adjacent structures.   Unimportant
features are the inflammatory processes which are often seen on the
inner and outer surface of the dura mater, and the hemorrhages in the
peridural cellular tissue.   Much more important are the changes in the
nerve roots and spinal cord.

The microscope shows hyperæmia of the nerve roots, infiltration of
the interstitial tissue with round cells, and degeneration of the nerve
fibres.

It is not astonishing that the spinal cord should take part in the in-
flammation, since the pia mater sends innumerable prolongations into
the interior of the cord.   In many cases, the cord, on transverse section,
appears diffluent, œdematous, and injected in places, in other parts ex-
tremely pale.   In not a few cases, changes are only observed upon
microscopic examination of sections of the hardened cord.   They are
sometimes very slight, and are partly parenchymatous, partly interstitial
in character.   In places, the axis cylinders are found swollen and un-
usually large; in addition, the medullary sheaths may be degenerated,
the neuroglia increased in places, and its nuclei proliferated.   The pro-
longations of the pia mater into the cord present distention of the vessels,
emigration of white blood-globules, and increase of the cellular elements.
In the gray matter the secondary changes are less marked than in the
white matter, but the ganglion cells are not infrequently swollen.

Atrophy and degeneration of the cord, particularly in the posterior
columns, have also been observed.

III. SYMPTOMS.—The manifest symptoms are not infrequently pre-
ceded by prodromata, such as anorexia, chilly sensations, general mal-
aise, etc.   The scene is sometimes opened by a single severe chill, followed
by fever, of no particular type, which may attain a considerable height
(40° C.).

The symptoms attributable to the meningitis itself are relatively
slight.   As the pia mater is very rich in nerves, we may attribute to in-
flammation of this membrane the pains and stiffness in the spinal
column, which increase on movement.   But, as a rule, these pains are not
increased by pressure on the spine, the passage of the cathode of a gal-
vanic current, etc.

All other symptoms are the results of irritation or paralysis of the
spinal nerves, or implication of the spinal cord itself.

Almost all patients complain of radiating pains in the limbs, chest, or
trunk, as the result of irritation of the posterior nerve roots.   The same
cause gives rise to the cincture feeling around the thorax or abdomen.

There is hyperæsthesia and hyperalgesia of the skin, so that very
severe pains are produced on gently touching or pricking the integument.
Muscular sensibility is also increased very greatly; the patients cry out
aloud when the muscles are carefully pressed between the fingers.

Muscular twitchings are frequent.   They are sometimes reflex, as
the result of previous pain, sometimes the result of pressure on the mus-
cles, or volitional movement, or they may be apparently spontaneous.

Stiffness of the muscles or contractures are observed not infrequently, the latter involving chiefly the extensors.

If the process extends to the cervical cord, the muscles of the back of the neck undergo tonic contracture (rigidity of the neck). The head is drawn more or less backwards, and the occiput is bored deep into the pillow. Active and passive movements of the head anteriorly, and sometimes rotatory movements, are impeded or entirely prevented. The entire body can often be lifted by the occiput like a rigid piece of wood.

Contracture of the muscles of the back is shown by the unusual anterior convexity of the spine, so that the trunk touches the bed only at the occiput and sacrum.

Contracture of the chest muscles interferes with inspiration; contracture of the abdominal muscles produces retraction of the abdomen.

Retention of urine and fæces is a common symptom, and is attributed to spasm of the vesical and rectal sphincters. The urine is generally scanty, dark in color, and contains a sediment.

Not infrequently there are changes in the pupils (contraction or inequality) as the result of disturbed innervation of the cilio-spinal centre.

Other symptoms, such as acceleration of the pulse and respiration, etc., depend upon the fever, rather than upon the meningitis.

The symptoms described must be regarded as irritative phenomena, and form the first stage of the disease. Recovery may occur, or the disease may progress to the second or paralytic stage. The transition is not sharply defined, and irritative and paralytic symptoms are often present at the same time. Paralytic symptoms must be looked for when the nerve roots are changed to such an extent as to render conduction impossible. Anæsthesia and analgesia take the place of hyperæsthesia and hyperalgesia. The muscles become paretic, then paralyzed, and show the signs of degeneration reaction. They undergo degenerative atrophy after the disease has lasted for some time. Reflex irritability is abolished. The function of the bladder is disturbed—at first retention, later incontinence. The urine is often unusually watery, pale, and abundant; it sometimes contains sugar.

These symptoms may also disappear. Indeed, recovery may occur in a comparatively short time, if the paralysis is the result of pressure, rather than of parenchymatous changes in the nerves. In many cases, permanent paralysis and atrophy of certain muscles are left over, or the signs of a chronic spinal affection make their appearance.

Great dangers may arise if the inflammatory process extends upwards and attacks the medulla oblongata. This is shown by difficulty in speech and deglutition, frequent vomiting, irregular respirations, which sometimes assume the Cheyne-Stokes type; the pulse, at first slow, becomes so rapid that it can no longer be counted, and the temperature often becomes hyperpyrexial. Death may result from suffocation, paralysis of the heart, or excessive rise of temperature.

The disease sometimes lasts only a few days, occasionally weeks and months; sequelæ may persist for life.

IV. DIAGNOSIS.—The diagnosis is usually easy. It is distinguished from rheumatism of the muscles of the back by the facts that, in the latter affection, severe and febrile constitutional symptoms are generally absent, that the muscles are tender on pressure, and that there are no muscular twitchings, sensory disturbances in remote parts or vesical symptoms. Meningitis with extensive, long-continued muscular twitchings may simulate tetanus, but the latter presents no sensory

changes and bladder symptoms, the muscles of mastication generally take part in the tonic contractions, and the contractions are increased by peripheral irritations. Finally, acute meningitis may be mistaken for acute myelitis. In myelitis the irritative symptoms are less prominent, while paralysis generally develops early. In myelitis the pains in the back are less and hyperæsthesia is generally absent, while anæsthesia develops very early; vesical paralysis and ammoniacal decomposition of the urine are soon produced; fever is slight or absent; trophic cutaneous changes are more frequent than in meningitis.

V. Prognosis.—The prognosis is always grave. The more prominent the paralytic symptoms the more dangerous is the situation. If symptoms of implication of the medulla oblongata or brain make their appearance, recovery is exceptional. It must also be remembered that permanent paralyses and atrophies are left over not infrequently, or that the signs of a chronic spinal affection finally develop.

VI. Treatment.—This is similar to that of acute myelitis (vide page 88).

4. *Chronic Spinal Meningitis.*

I. Etiology.—Chronic spinal meningitis or leptomeningitis either develops as such from the start, or it forms the terminal stage of an acute attack. The latter is apt to occur if there are repeated exacerbations of the inflammation in rapid succession, or if injurious influences exercise their effect during the stage of convalescence.

The etiology is, in part, the same as that of acute meningitis, but certain etiological peculiarities must be taken into consideration. Thus, there can be no doubt that certain cases are produced by alcoholic excesses. Chronic meningitis also occurs in syphilis and leprosy. According to Koehler, chronic diseases of the lungs, heart, or liver predispose to the disease by producing circulatory stasis. Finally, it often, indeed almost constantly, accompanies many chronic diseases of the cord; for example, tabes dorsalis, multiple sclerosis, etc. Most authors regard the meningitis, under such circumstances, as a sequel and complication of the chronic spinal affection, while others consider the meningitis primary, the spinal disease secondary.

II. Anatomical Changes.—The lesions rarely extend over the entire length of the cord, but are generally confined to more or less extensive, often multiple foci. The lower portions are most frequently, the upper cervical region rarely, affected. The changes are usually more marked on the posterior surface.

Chronic meningitis is characterized by thickening and fibrous opacities of the pia mater and arachnoid. The new-formed connective tissue may attain a thickness of several millimetres, and feel as hard as cartilage. Calcification and ossification also occur, particularly in the arachnoid tissue.

In many cases the inflamed parts are unusually filled with blood, particularly in the veins and capillaries. There may also be an abnormal production of pigment, resulting in brownish-red or black patches (secondary to previous hemorrhages). It must be remembered that pigmentation of the meninges, particularly in the cervical region, occurs in healthy individuals in old age.

There are numerous, sometimes very extensive adhesions to the dura mater. The spinal fluid is often excessive in amount, and is not infre-

quently cloudy and mixed with flocculi. In rare cases it assumes a more purulent character.

On making a transverse section of the cord, it is sometimes seen that the prolongations of the pia mater into the substance of the spinal cord are unusually broad. The pia mater becomes so adherent to the cord that it cannot be detached from the latter without producing losses of substance.

The dura mater presents thickening, calcification, and adhesions, and its inner surface is often nodular and granular. The microscope reveals connective-tissue proliferations which contain chalky concretions.

The nerve roots are often flat and atrophic, the result of compression by the connective-tissue new-formations. Internally they present inflammatory processes, and the microscope not infrequently shows degenerative changes in the nerve fibres.

The spinal cord may contain macroscopic or microscopic foci of disease—annular sclerosis, ascending and descending secondary degeneration, destruction of nerve fibres, interstitial proliferation of connective tissue in spots, etc.

III. SYMPTOMS.—The symptoms are similar in many respects to those of acute meningitis, but they are less violent, more often present remissions and exacerbations, and are unattended with fever, unless accompanied by some febrile complication. We will merely point out that the symptoms are irritative or paralytic, or a combination of the two.

There are frequent complaints of pain and stiffness in the spine, and of rigidity of the back of the neck, if the disease is situated sufficiently high. The pains increase on motion, but not on pressure upon the spine. Complaint is often made of a cincture feeling and radiating pains in the limbs. Hyperæsthesia and paræsthesiæ are often noticed in the beginning, and are followed at a later period by anæsthesia, which is usually incomplete. G. Fischer observed polyæsthesia in a number of cases. Tonic or clonic muscular twitchings or contractures occur very commonly, and micturition and defecation are often delayed. If the compression of the nerve roots increases or their nerve fibres degenerate, paralytic symptoms will become prominent. They appear more frequently as paresis than paralysis, and often increase in dorsal decubitus, in other cases during the erect position. They are followed by degenerative atrophy of the paralyzed muscles, degeneration reaction, and abolition of reflex irritability. Paralysis of the bladder and rectum, and decubitus will ensue if the lower spinal nerves or the cord itself are implicated.

The disease sometimes lasts for years. It may terminate in recovery, or permanent paralyses and atrophy remain, or acute fatal exacerbations develop, or the process extends to the medulla oblongata, or death is the result of decubitus, or of cystitis and ammoniacal decomposition of urine. Chvostek claims that the calcareous plates mentioned above (but whose inflammatory origin is not always certain) may be the source of very obstinate pains.

IV. DIAGNOSIS.—The diagnosis is sometimes very difficult, and if it is associated with an affection of the cord itself, it will be almost impossible to differentiate the symptoms of both diseases. In general, the principles laid down in the differential diagnosis of myelitis hold good here. The onset of the malady may be mistaken for tabes, but the latter presents no paralytic symptoms, pupillary changes are generally noticed, the anæsthesia is more marked, the patellar reflex disappears early,

the radiating pains are more violent, and ataxic symptoms soon appear. Spinal irritation, for which the disease may also be mistaken, affects anæmic, hysterical, nervous individuals, paralytic symptoms are absent, the spine is tender on pressure, and there is a great disproportion between the subjective complaints and objective changes.

V. Prognosis.—The prognosis depends on the same factors as that of acute spinal meningitis, and this is also true of treatment.

5. *Meningeal Apoplexy.*

(*Hæmatorrhachis.*)

I. Anatomical Changes.—Meningeal hemorrhages occur most frequently into the loose peridural cellular tissue which separates the dura mater from the bony spinal canal. They fill the epidural space (Fig.

Fig. 86.
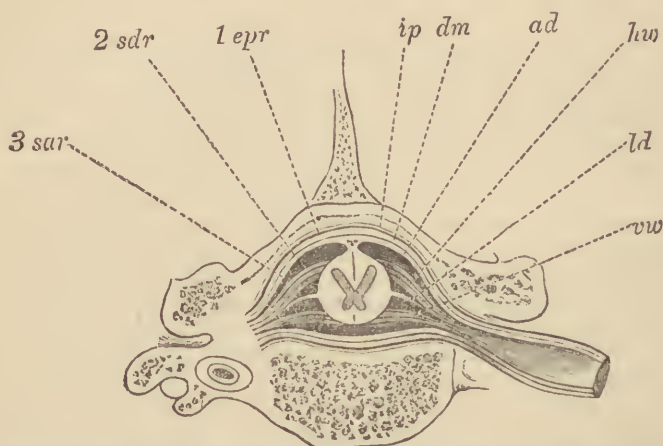
Transverse section through the vertebral column and spinal cord (schematic).

ip, internal periosteum of the vertebræ ; *dm*, dura mater ; *ad*, arachnoid ; *hw*, posterior nerve root ; *ld*, ligamentum denticulatum ; *vw*, anterior nerve root ; 1 *epr*, epidural space ; 2 *sdr*, subdural space ; 3 *sar*, subarachnoid space.

86, 1 *epr*), and are known as epidural apoplexy. A second site is the subdural space (Fig. 86, 2 *sdr*), between the dura and arachnoid (subdural or arachnoidal apoplexy). Finally, hemorrhages may occur in the meshed tissue between the arachnoid and pia mater, the subarachnoid space (Fig. 86, 3 *sar*), and are known as subarachnoid apoplexy.

In epidural apoplexy, the hemorrhage rarely extends along the entire cord, but only over a few vertebræ, or occurs in several scattered foci. The hemorrhage is sometimes confined to the posterior surface, or also extends to the sides; it rarely involves the entire circumference of the cord. The blood generally forms dark-red, loose clots, which may be so extensive as to push the dura inwards and compress the cord. The dura itself, or its outer surface, may present bloody suffusions, and extravasions are also observed in the nerve roots. The vessels of the dura mater are often congested. Secondary changes are slight or entirely absent.

The clinical symptoms indicate that these lesions are capable of re-

covery.   The blood-clots are probably absorbed as in other organs, and deposits of pigment and adhesions are ge erally left over.   In especially unfavorable cases there may be irreparable atrophy of some of the nerve roots and grave changes in the substance of the spinal cord.

The changes in subdural apoplexy are the same as in internal hemorrhagic pachymeningitis (vide page 166).

In subarachnoid apoplexy, the amount of blood may be very considerable.   It is sometimes derived from hemorrhages within the skull, and may extend along the entire length of the cord.   The spinal cord and nerve roots are not infrequently compressed.

II. ETIOLOGY.—Meningeal apoplexy is one of the rarer diseases, and is more common in men than in women.   In some cases no cause can be discovered.   Not infrequently it is attributed to injury (incised and gunshot wounds, fall, blow or violent concussion of the spine, lifting heavy loads, instrumental extraction of the new-born).

In some cases the hemorrhage is associated with diseases of adjacent organs (tuberculosis or cancer of the spine, rupture of aortic aneurism into the spinal canal, acute inflammations of the cord and its meninges).

It is sometimes the result of excessive active or passive congestion of the meninges.   This occurs in suppressio mensium, or suppressed hemorrhoidal hemorrhages, in cardiac hypertrophy, violent excitement; in those who have died of tetanus, eclampsia, epilepsy, chorea, hydrophobia or asphyxia; in toxic spasms; in chronic diseases of the heart, lungs and liver.

Meningeal hemorrhages sometimes occur in infectious diseases, as the result of so-called blood dissolution or the hemorrhagic diathesis.

Finally, they may be derived from a cerebral hemorrhage which has flown into the spinal canal, or from rupture of the vertebral or spinal artery.

It is not known whether the hemorrhage is ever the result of primary affections of the vessels.

III. SYMPTOMS.—The symptoms are characterizd by their sudden onset and apyrexial course.   Prodromata are rare and result from congestion of the cerebral and spinal meninges (slight pains in the back, a feeling of heaviness, etc.).   Slight febrile movement sometimes occurs on the second or third day as the result of reactive inflammation.

Consciousness almost always remains intact, unless the hemorrhage is situated very high, or is very large in amount.

The patients suddenly complain of pain in a certain part of the spine. The pains often extend along the entire spine, shoot into the arms, chest, abdomen, or legs, according to the site of the hemorrhage, and give rise to a cincture feeling around the chest or belly.   The spine is not tender, but there is a feeling of stiffness, and even rigidity of the back of the neck if the hemorrhage is situated high up.   These symptoms result in part from irritation of the meninges, in part from irritation of the nerve roots.

Irritative and paralytic symptoms may be closely connected in the further course of the disease.   The former include hyperæsthesia of the skin, paræsthesia, tonic and clonic muscular twitchings and contractures; the latter include partial, rarely complete, anæsthesia and motor paralysis.   The paralyses are often slight at first, but increase at the end of a few hours.   In other cases, the paralysis improves very rapidly. Atrophy of the muscles, degeneration reaction, and abolition of reflex

excitability may be expected if the nerve roots are compressed for a long time. If the substance of the spinal cord is affected, paralysis of the bladder and rectum, cystitis, and decubitus may develop.

As a rule, the disease lasts from two to eight weeks, but in not a few cases the duration is much longer. Death sometimes occurs, with symptoms of shock, very soon after the beginning of the hemorrhage. In other cases, the hemorrhage is situated so high that death results from disturbances of innervation in the medulla oblongata. The disease is sometimes complicated with meningitis, and the extension of the latter to the medulla oblongata causes the fatal termination. Recovery is often incomplete, and permanent paralysis and atrophy of groups of muscles are left over.

IV. DIAGNOSIS.—The diagnosis is generally easy; special importance must be attached to the apoplectiform beginning of the symptoms, and their apyrexial course. In acute spinal meningitis, there is febrile movement, the irritative symptoms are more intense, and the symptoms develop more gradually. In acute myelitis and in hemorrhage into the cord, the pains are slight or absent, paralytic symptoms predominate, and there is a rapid development of trophic disturbances, decubitus, paralysis of the bladder, and decomposition of the urine. In concussion of the spine, also, the irritative symptoms are less marked, paralytic symptoms are prominent from the beginning.

The localization of the hemorrhage is not difficult. If the lumbar cord is affected, the symptoms refer chiefly to the lower limbs, bladder, and perineal region, and priapism is occasionally observed; hemorrhages into the dorsal region are attended by a cincture feeling around the abdomen or thorax; cervical hemorrhages are attended by irritative and paralytic symptoms in the upper limbs; hemorrhages in still higher regions are followed by bulbar symptoms. In addition, the localization of the pain in the spine must be taken into consideration. Whether the hemorrhage is epidural, subdural, or subarachnoidal depends particularly on the etiology.

V. PROGNOSIS.—A relatively large number of cases terminate in complete, or almost complete, recovery. The situation is so much more serious the nearer the hemorrhage is to the medulla oblongata, the larger it is, and the more the nerve roots and cord are compressed.

VI. TREATMENT.—This is similar to that of hemorrhages into the substance of the spinal cord (vide page 81).

6. *Tumors of the Spinal Meninges.*

I. ANATOMICAL CHANGES.—These tumors may be epidural, subdural, or subarachnoidal, but clinically their symptoms depend merely on irritation of the meninges and of the substance of the cord or paralysis of these parts by excessive compression. Tumors which measure more than ten centimetres in length or four to five centimetres in thickness are exceedingly rare. Further growth is only possible by proliferation through the intervertebral foramina and continued growth outside of the spinal canal, or by implication of the bodies of the vertebræ in the neoplastic process.

Meningeal tumors start most frequently from the dura mater, and are situated either upon its outer surface or project from its inner surface into the subdural space; they start less frequently from the pia mater or arachnoid tissue. The meninges in the vicinity are often inflamed. The growths are generally round or oval in shape, and often bore a hole into the substance of the cord (vide Figs. 87 and 88). They are almost always single, rarely multiple. In some cases, tumors are found only on the spinal meninges, in others they are also present in the cranial cavity, the spine, adjacent organs, or remote parts of the body.

Fibroma, lipoma, myxoma, sarcoma, psammoma, melanoma, neuroma, tubercle, gumma, enchondroma, osteoma, and carcinoma are the varieties of meningeal tumors which have been described.

Cancer of the meninges is rarely primary, but usually extends from adjacent parts or spreads by metastasis. Closely allied to neoplasms are the animal parasites, viz., echinococci and cysticercus cellulosæ (in one case reported by Westphal).

Circumscribed foci of inflammation and hemorrhage of the meninges are coordinate with tumors from a clinical standpoint.

After the neoplasms have attained a certain size, they compress the nerve roots and spinal cord. At first the nerve roots are swollen, succulent, and reddened; later they become gray and atrophic. Compression of the cord rarely gives rise to simple circular atrophy, but, as a general thing, to compression myelitis which is especially apt to progress below the site of compression, and not infrequently gives rise to secondary degeneration. In rare cases, there is purulent liquefaction of the lower portion of the cord.

II. ETIOLOGY.—Meningeal tumors occur most frequently in males in middle life. Their causes are generally unknown. They are sometimes attributed to injury and cold. According to some authors, the tumors are apt to develop or increase rapidly during pregnancy and the puerperal condition. Leyden con-

Fig. 87.   Fig. 88.

Anterior view.   Compression of the cord visible after removal of the tumor.
Tumor growing from the spinal dura mater. After Charcot.

siders mental excitement, especially fright, as a possible cause. Tumors are hereditary in some families.

III. SYMPTOMS.—As a rule, symptoms do not appear until the nerve roots or spinal cord are affected. They are sometimes preceded for years by peculiar painful sensations in certain parts of the spine, which may increase on making certain movements, and are probably the result of local irritation by the tumor. Some portions of the spine may be tender on pressure.

The irritative symptoms increase in severity when the tumor begins to grow. The patients complain of a cincture feeling, and suffer from radiating pains in the limbs, cutaneous hyperæsthesia and paræsthesiæ; tonic and clonic muscular twitchings and contractures make their appearance.

The stage of irritation passes more or less suddenly into that of paralysis, characterized by anæsthesia, and paresis or paralysis of the muscles. If these symptoms depend on compression and subsequent degeneration of the nerve roots, atrophy of the muscles, degeneration reaction, and loss of reflex excitability will soon make their appearance.

In other cases, the paralytic symptoms indicate implication of the cord. If the tumor starts from the anterior surface of the meninges, muscular paralysis will occur very early; if it starts from the posterior surface, sensory disturbances will be more prominent.

In some cases, the compression·is confined to one-half of the cord, and the symptoms of a unilateral lesion are produced, *i. e.*, paralysis and hyperæsthesia on the side of the compression, anæsthesia on the other side (vide page 138).

If the tumor simply interrupts conduction in a transverse section of the cord, paraplegia, anæsthesia, paralysis of the bladder and rectum, increase of the tendon reflexes and of reflex excitability are noticeable.

If simple compression is followed by compression myelitis which extends deep into the lumbar cord, reflex irritability becomes lost, the paralyzed muscles atrophy, and lose their electrical excitability.

Descending degeneration of the pyramid tracts may be suspected if muscular contracture and twitchings set in and the tendon reflexes are markedly increased.

As a matter of course, the clinical history may vary from time to time.

As a rule, the disease progresses uninterruptedly to an unfavorable termination. Recovery is only possible in the case of inflammatory and syphilitic products. The disease may last months or years. Death is sometimes the result of marasmus, or is brought about by the primary tumor in other organs. In some cases, it results from decubitus or cystitis and ammoniacal decomposition of the urine. Acute meningitis sometimes develops and proves fatal. If the tumors are situated very high, bulbar symptoms may be produced and terminate in death.

IV. DIAGNOSIS.—No single symptom is positive evidence of a meningeal tumor. The same symptoms also occur in other conditions. We infer that they are produced by tumors when the presence of new growths may be demonstrated in other organs or in the vicinity of the spinal column, or when the patient is scrofulous, phthisical, or syphilitic. If these diagnostic aids are wanting, we must suspect a meningeal tumor if there are symptoms of increasing compression of the cord, which start in a circumscribed region.

The diagnosis of the level of the tumor is usually easy. Sometimes it is evident from visible changes in the spine; in other cases the patients feel pain in this spot, or the spine is tender over certain parts. In addition, we are aided by the situation of the cincture feeling, and the distribution of the eccentric pains, sensory disturbances, and paralyses.

In tumors of the cauda equina, the patients complain of the most violent pains in the sacral region, anæsthesia develops, with paralysis and atrophy of the muscles, abolition of electrical and reflex excitability, and the bladder and rectum are often paralyzed. True spinal symptoms (motor and sensory paraplegia) are absent, and the symptoms occur only in the distribution of certain peripheral nerves.

V. PROGNOSIS.—Recovery is possible in those tumors alone which are the result of syphilis.

VI. TREATMENT.—The treatment is similar to that of intramedullary tumors.

PART III.

DISEASES OF THE MEDULLA OBLONGATA.

1. *Progressive Bulbar Paralysis.   Glosso-labio-laryngeal Paralysis.*

I. ETIOLOGY.—Progressive bulbar paralysis depends on disappearance of the large ganglion cells of the nerve nuclei situated in the floor of the fourth ventricle. These cells correspond in function and anatomical arrangement to the large ganglion cells in the anterior horns of the spinal cord.

The disease is not very frequent, nor is it exceedingly rare. It is more common in men than in women, and from the age of forty to sixty years; it is also somewhat more frequent among the better classes.

Colds and injuries of all kinds are often mentioned as direct causes. Stein described a case as the result of strain from protracted blowing of wind instruments. It has also been attributed to mental excitement.

The influence of heredity has not been proven with certainty. Syphilis is mentioned as a cause in a number of cases. The first symptoms sometimes develop during convalescence from infectious diseases.

Progressive bulbar paralysis may be primary or secondary. The former variety develops independently, not infrequently extends secondarily to the spinal cord, and gives rise to the symptoms of spinal progressive muscular atrophy. The secondary form follows progressive muscular atrophy or amyotrophic lateral sclerosis.

II. SYMPTOMS.—As a rule, the disease begins gradually, and its further progress is also slow but uninterrupted. In very rare cases, it has a sort of apoplectiform beginning, as in Kussmaul's patient, who suddenly noticed difficulty in speech while preaching. The prodromata generally consist of tearing pains in the back of the neck and occiput, a feeling of constriction in the throat, and dizziness. In one of Leyden's cases the disease began with attacks of dyspnœa.

The first manifest symptom is difficulty in the movement of the tongue, shown by subjective and objective changes in speech and mastication. The patients quickly grow tired from talking or chewing, and articulation becomes indistinct.

Difficulty in movement and visible atrophy of the lips (paralysis and atrophy of the orbicularis oris) gradually develop. Then symptoms occur which must be attributed to paralysis of the muscles of the palate, pharynx, and larynx.

All the symptoms are bilateral. Bamberger has recently reported a case, however, in which the affection appeared to be unilateral, but no autopsy could be obtained.

In rare cases, the disease begins in the lips. It almost always advances uninterruptedly, though slight remissions and exacerbations sometimes occur. In one of his patients, Kussmaul noticed the exacerbations at the period of menstruation.

The paralysis of the tongue begins as mere impairment of motion, but gradually passes into complete paralysis. The tongue then can no longer be moved in any direction, and lies on the floor of the mouth like a lifeless mass; the backward movements of the organ sometimes retain considerable vigor.

In many cases the tongue undergoes atrophy. Its surface is furrowed and wrinkled, its volume is diminished, and, on attempting to protrude it, active fibrillary contractions become visible.

At the same time, the difficulties in speaking and eating become more marked, and increase still more when the other groups of muscles mentioned above become paralyzed. Among the vowels, the articulation of i is affected most frequently, because this requires the approximation of the dorsum of the tongue to the hard palate. Among the consonants b and sch are the ones which are first lost, then s, l, k, g, t in succession, and finally d and n. All these disturbances are purely mechanical (anarthria or paralytic alalia).

The paralysis of the tongue also interferes with the formation of a bolus and with deglutition. The tongue cannot form the food into a bolus or remove particles which have passed between the gums and cheek. The patient is often compelled, by the aid of the finger or a spoon, to bring the food back into the buccal cavity. Since the tongue, by its apposition against the hard palate, must force the bolus backwards, paralysis of the tongue itself suffices to produce disturbances of deglutition. Some patients push the bolus backwards with the fingers, etc., or bend the head strongly backwards so that the bolus may fall backwards from the action of gravity.

If the orbicularis oris becomes paralyzed, the difficulties in articulation and mastication increase. Among the vowels, the pronunciation of o and u is first rendered impossible because it necessitates the puckering of the mouth; later i and e disappear, while a is retained so long as phonation is at all possible. The

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consonants are affected in the following order: at first, p and f, then k and m, finally w.

As a result of the paralysis of the lips, the mouth is constantly kept more or less open, so that fluid or solid food is apt to regurgitate in part from the mouth.

This paralysis is also one of the causes of the salivation from which the patients suffer almost constantly. In part, however, this symptom is the result of the disturbance of deglutition. The salivation generally ceases spontaneously at night, when the patients assume the dorsal decubitus, and the saliva may flow backwards. In some cases, the amount of saliva has been found to be increased, probably as the result of irritation of the centres of salivary secretion situated in the medulla oblongata. Some authors regard the salivation as paralytic in its origin—it occurs after section of the chorda tympani nerve—but the increase in

Fig. 89.

Facial expression in progressive bulbar paralysis. After Leyden.

the amount of saliva is sometimes too large to be explained in this manner. In many patients, the saliva flows almost incessantly out of both angles of the mouth, and this may give rise to erythema of the chin.

Atrophy of the paralyzed lips is often evident upon feeling them between the thumb and index finger. The patients are unable to pucker the lips in order to kiss, blow, etc. The mouth appears to be widened; the entire expression of the face is changed, particularly if other facial muscles have undergone atrophy (levator anguli oris, quadratus menti, buccinator, etc.). The frontal branches of the facial nerve are usually unaffected, so that the mobile forehead contrasts very strongly with the mask-like, lower half of the face with its tearful expression. The face also manifests an expression of astonishment (vide Fig. 89).

Paralysis of the palatal muscles increases the disturbances in articulation and deglutition. Speech becomes nasal, and the enunciation of b and p becomes im-

possible if a sufficiently large current of air may escape through the nose, on account of imperfect closure of the naso-pharyngeal cavity. *B* and *p* then sound like *me*, *we*, or *fe*, but resume their normal acoustic character if both nostrils are closed with the fingers and the escape of air through the nose is thus prevented. The patients often swallow the wrong way. On inspection, the palatal arch appears to be flaccid, sometimes more dependent on one side than on the other, moves very little or not at all during phonation, and flutters to and fro during vigorous respiratory movements.

Speech gradually degenerates into an unintelligible grunting, so that the patients must express themselves in writing or, if the arms are paralyzed, by making signs with the eyes or movements of the head. On account of paralysis of the vocal cords and patency of the glottis during phonation, the latter act requires the expenditure of a large amount of air and vigorous action of the abdominal muscles, so that the patients rapidly grow tired.

Paralysis of the organs of deglutition is shown by the increasing disturbances during the act of deglutition. The bolus often remains in the pharynx or œsophagus, and puts the patient in danger of suffocation. After a while, the food accumulates, during deglutition, between the anterior surface of the epiglottis and base of the tongue in the pyriform sinuses, and distends them to such an extent as to interfere with respiration. Many patients must be nourished entirely with the œsophageal sound.

Paralysis of the muscles of the larynx interferes with deglutition, inasmuch as the food may enter the larynx, on account of its imperfect closure. This may be the source of foreign-body pneumonia. If closure of the larynx is impaired, on account of paralysis of the depressor epiglottidis, bits of solid food are especially apt to enter the larynx. A second form of closure of the larynx is produced during deglutition by the approximation of the arytænoid cartilages and ary-epiglottic folds. If this is impaired, fluids are particularly apt to enter the larynx.

The laryngoscope often shows impaired mobility and diminished tension of the vocal cords, or even complete paralysis of the recurrent laryngeals.

Paralysis of the muscular coat of the bronchi is also said to occur, and to be manifested by difficulty in yawning, coughing, hawking, etc.

The paralyses described above occur in the distribution of the hypoglossal, glosso-pharyngeal, pneumogastric, spinal accessory, and facial nerves. Other cerebral nerves are rarely affected. In one case, Hérard described paralysis of the abducens nerve. Paralysis of the motor branches of the trigeminus has also been reported.

The muscles of the back of the neck are often atrophied, so that the head sinks over forwards and can be rotated very slightly or not at all. The muscles of the thenar and hypothenar eminences, and the interossei often undergo atrophy and paralysis, and this may be followed by the extensive spread of the disease as in spinal progressive muscular atrophy. Finally the patients may become entirely helpless and incapable of motion. Mental disturbances have not been observed hitherto. At the most, the mood is tearful or laughing without reason.

The paralyzed muscles and nerves present partial degeneration reaction, as in progressive muscular atrophy (vide page 127).

The mechanical excitability of the muscles has also been found increased. The occurrence of diplegic contractions possesses no special significance.

Cutaneous sensibility is always unchanged. Some patients complain of pains in the neck and occiput, and also in the back and limbs if progressive muscular atrophy develops.

The reflex irritability of the mucous membrane of the mouth, pharynx, and larynx is often abolished or greatly diminished. Krishaber noticed this as one of the first symptoms in two cases.

The disease runs a chronic course, the average duration being one to three years. In one of Leyden's cases it lasted seven years. Death may

be the result of increasing marasmus from defective nutrition, of "foreign-body" pneumonia, of attacks of dyspnœa or sudden heart failure from paralysis of the pneumogastric and spinal accessory. In Blumenthal's case the dangerous dyspnœa did not subside until tracheotomy had been performed.

Fatal attacks of syncope also develop in some cases. We must not forget to mention that paralysis of the pneumogastric and spinal accessory is also shown by the constant or paroxysmal increase in the frequency of the pulse (to one hundred and fifty per minute). This is sometimes preceded by retardation of the pulse.

III. ANATOMICAL CHANGES.—The anatomical changes affect the nerve nuclei of the floor of the fourth ventricle, and the corresponding nerves and muscles. The sympathetic and the spinal ganglia have been found intact.

The changes in the bulbar nuclei are exactly like those found in the

Transverse section through the upper part of the normal medulla oblongata. *hk*, hypoglossal nucleus. Enlarged 25 times.

The same in progressive bulbar paralysis. *hk*, hypoglossal nucleus, almost destitute of ganglion cells. After Leyden.

anterior horns of the cord in spinal progressive muscular atrophy. The large ganglion cells atrophy and disappear, generally as the result of yellow pigment degeneration. The cells are filled more and more with golden yellow, granular pigment, the nuclei are at first concealed and then disappear, the cells become smaller and smaller, lose their processes, and finally form small round clumps of pigment, which may also undergo absorption (vide Figs. 90 and 91).

Whether the process is a simple degenerative atrophy or a primary inflammatory change in the ganglion cells, or whether the ganglion cells are affected secondarily to inflammatory interstitial processes, is still unsettled. In our opinion the disease, like progressive muscular atrophy, is a primary degenerative atrophy of the ganglion cells.

Macroscopically, the medulla may appear intact, although flattening, narrowing, and changes in the consistence of the organ have also been described. It may also appear intact to the naked eye, after hardening

in chromic acid or chromates, although extensive changes are found under the microscope.

The emerging nerve roots are also atrophied and degenerated, so that, on transverse section through the medulla oblongata, their fibres are found atrophied or have disappeared (vide Fig. 92).

The process affects adjacent nerve nuclei. It begins almost always in the nuclei of the hypoglossal nerve, which are situated in the lower half of the medulla, next to the median line (Fig. 93, 4 *hk*). The nuclei of the spinal accessory and pneumogastric are immediately adjacent (Fig. 93, 6 *vk*, and 7 *acck*), and are soon involved in the process. But the nucleus of the acoustic nerve (Fig. 93, 5 *ak*) almost always escapes, so that auditory disturbances constitute very rare symptoms. The facial is involved very early, although its nucleus is situated in the upper half of

FIG. 92.

Transverse section through the medulla oblongata in progressive bulbar paralysis ; *hk*, hypoglossal nucleus, with disappearance of the ganglion cells ; *vv*, atrophied remains of the root bundles of the hypoglossus ; *py*, pyramid tract ; *ov*, olivary body. Enlarged 6 times. After Leyden.

the medulla (Fig. 93, 2 *fa*). According to many writers, however, there is also another facial nucleus in the lower half of the medulla, about at the level of the nuclei of the hypoglossus and spinal accessory nerves. The implication of the glosso-pharyngeal nerve is not astonishing, since its nucleus is situated deep in the medulla, immediately adjacent to that of the pneumogastric. The nucleus of the trigeminus, corresponding to the locus cœruleus (Fig. 93, 1 *lc*), is so far from the main site of the morbid process that it usually escapes, and hence disturbances in mastication are rare in this disease. Implication of the abducens may be expected more frequently, since its nucleus is very close to the root fibres

of the facial nerve and, immediately adjacent to the knee of the facial fibres, corresponds to the eminentia teres (Fig. 93, 3 *et*).

Fig. 94 shows a transverse section of the medulla oblongata, corresponding to about the middle of the olivary bodies; Fig. 95 shows the points of exit of the different nerves.

The changes within the medulla oblongata are not always confined to the nerve nuclei and the corresponding nerves. The surrounding parts present increase of the interstitial connective tissue, accumulation of granulo-fatty cells and amyloid bodies, and degenerative atrophy of the nerve fibres. In the walls of the blood-vessels, we sometimes noticed thickening, nuclear proliferation, fatty degeneration, and accumulation of fat granules, granulo-fatty cells and round cells in the lymphatic sheaths. Disease of the formatio reticularis and olivary bodies has been described, but particularly changes in the pyramid tracts. In one case, Leyden found changes in the latter extending upwards into the pons and downwards into the decussated and non-decussated pyramid tracts in the cord. The restiform bodies always remain intact.

The nerves in question (hypoglossus, spinal accessory, pneumo-

FIG. 93.
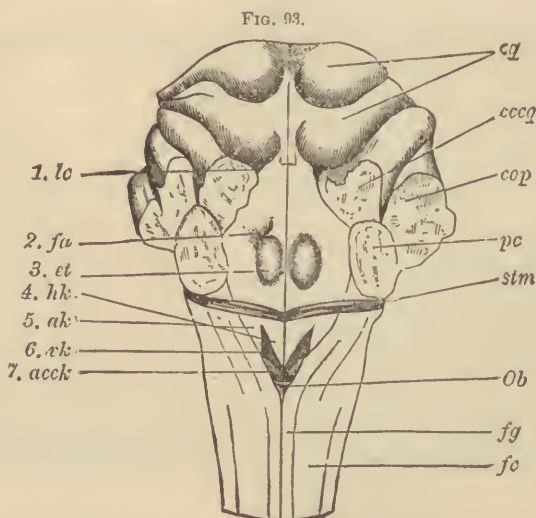
Posterior surface of the medulla oblongata, showing the fourth ventricle and the nuclei on its floor: 1 *lc*, locus cœruleus (nucleus of the trigeminus) ; *2 fa*, fovea anterior (facial nucleus) ; 3 *et*, eminentia teres (knee of the facial nerve) ; 4 *hk*, hypoglossal nucleus ; 5 *ak*, median nucleus of the posterior root of the acoustic nerve ; 6 *vk*, pneumogastric nucleus (ala cinerea) ; 7 *acck*, nucleus of the spinal accessory ; *cq*, corpora quadrigemina ; *cccq*, crura cerebelli ad corpora quadrigemina ; *cop*, crura cerebelli ad pontem ; *pc*, pedunculus cerebelli ; *stm*, striæ acusticæ ; *ob*, obex ; *fg*, funiculus gracilis ; *fc*, funiculus cuneiformis.

gastric, facial, glosso-pharyngeal, sometimes the abducens and motor branch of the trigeminus) are thin, flattened, gray, and transparent. Under the microscope, some of them are found to consist chiefly of connective tissue, while the nerve fibres have disappeared more or less completely (degenerative atrophy).

The muscles of the tongue and face also present degenerative changes: diminution in the size of the fibres, increase of the nuclei, disappearance of the muscular substance, proliferation of the interstitial connective tissue and its nuclei. The development of fat is sometimes so abundant that the muscles seem to have increased in size. Waxy degeneration has also been observed. Despite all these changes, the mus-

cles may appear unchanged to the naked eye. In advanced cases, they
have a light-yellow to butter-yellow color.

If bulbar paralysis is complicated with progressive muscular atrophy
or *vice versa*, atrophy of the large ganglion cells of the anterior horns of
the cord will also be noticed. If bulbar paralysis complicates amyotrophic lateral sclerosis, we will find a bilateral affection of the pyramid
tracts, degenerative atrophy of the ganglion cells in the anterior horns
of the spinal cord, and bulbar changes (vide page 135).

IV. DIAGNOSIS.—The diagnosis is easy. As a matter of course,
bulbar symptoms may develop, in an acute or chronic manner, in all possible diseases of the medulla oblongata, but their mode of development
will then be different. It will also be easy, as a general thing, to differentiate between primary or secondary progressive bulbar paralysis.

The disease must be differentiated from: *a. Hemorrhages, thrombosis,
and embolism* in the medulla oblongata; the latter generally begin suddenly, the paralysis is often mainly or exclusively unilateral, sensory

FIG. 94.
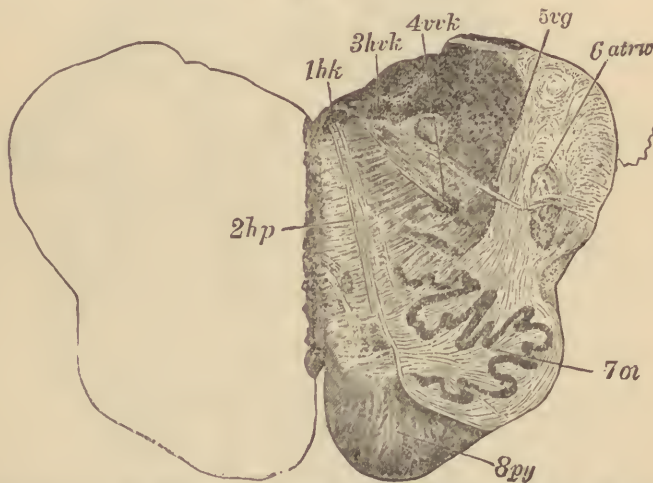
Transverse section through the medulla oblongata, corresponding to about the middle of the
olivary bodies. Enlarged 3 times. 1 *hk*, hypoglossal nucleus ; 2 *hp*, bands of fibres of the hypoglossus ; 3 *hvk*, posterior pneumogastric nucleus ; 4 *vvk*, anterior pneumogastric nucleus ;
5 *vg*, bands of fibres of the pneumogastric ; 6 *atrw*, ascending root of the trigeminus ; 7 *ov*,
olivary body ; 8 *py*, pyramid.

disturbances are frequent, there is absence of atrophy and of changes in
electrical excitability.

*b. Tumors which compress the medulla oblongata and emerging
nerves;* these often produce unilateral, or mainly unilateral paralytic
symptoms, often affect the trigeminus and acoustic nerves, produce
twitchings in the facial muscles and tongue, and complete degeneration
reaction in the paralyzed muscles; convulsions, vomiting, vertigo, choked
disc, and amaurosis are frequent symptoms.

c. Lesions of certain parts of the cerebrum sometimes give rise to
bulbar symptoms, but they are then unequal on the two sides; the electrical irritability is retained, the muscles do not undergo atrophy, and
the limbs are paralyzed.

d. Jolly described a case of *multiple cerebro-spinal sclerosis* with

marked symptoms of bulbar paralysis, but in which the medulla oblongata appeared to be intact, on autopsy. In cases of this kind, other symptoms are present in addition to the bulbar symptoms.

e. The disease will be mistaken for *diplegia facialis* of peripheral origin only on superficial examination. In such cases, all the branches of the facial nerve are paralyzed, and there is complete degeneration reaction. The muscles of the tongue, palate, and larynx are not paralyzed.

V. PROGNOSIS.—The prognosis is unfavorable, since the disease ad-

FIG. 95.
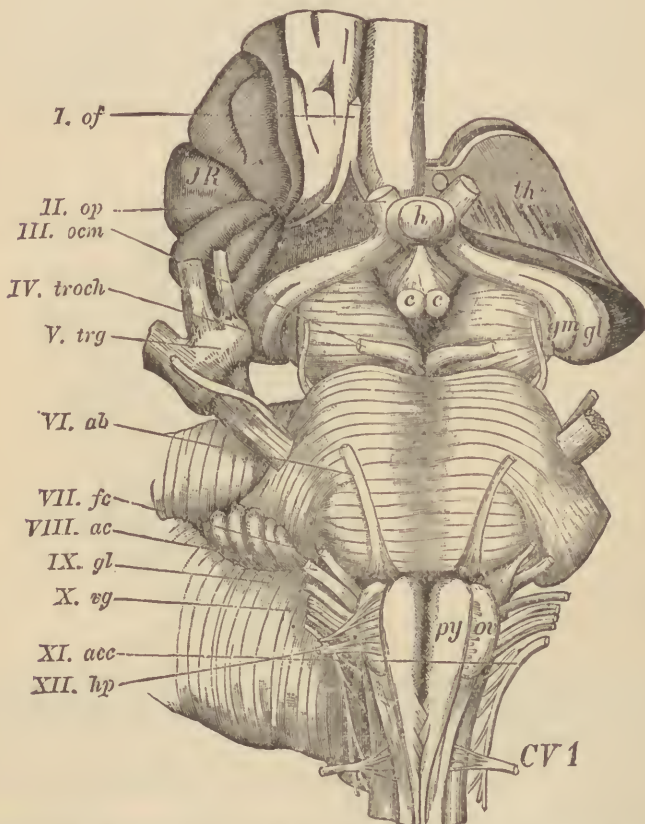
Medulla oblongata and adjacent portions of the brain, with emerging cerebral nerves. After Schwalbe. I. *of*, olfactory nerve: II. *op*, optic nerve; III. *ocm*, oculo-motor nerve; IV. *troch.*, trochlear nerve; V. *trg*, trigeminus nerve: VI. *ab*, abducens nerve; VII. *fc*, facial nerve; VIII. *ac*, acoustic nerve; IX. *gl*, glosso pharyngeus nerve; X. *vg*, pneumogastric nerve; XI. *acc*, spinal accessory nerve: XII. *hp*, hypoglossus nerve; *py*, pyramids; *ov*, olivary body; *th*, optic thalamus; *gmgl*, corpus geniculatum medium et laterale; *h*, hypophysis; *cc*, corpora candicantia. JR, Island of Reil.

vances uninterruptedly to a fatal termination. A few cases of improvement and even recovery have been reported, but the correctness of the diagnosis is questionable. Improvement is said to have been more apt to occur in syphilitic cases, but even these are doubtful.

VI. TREATMENT.—The regimen is not unimportant in treatment:

pure air, nutritious food, avoidance of stimulating food and drink, later careful feeding with the œsophageal sound.

At the beginning of the disease, derivatives may be applied high up on the back of the neck.

Not much can be expected from internal remedies (potassium iodide, nitrate of silver, arsenic, strychnine, etc.).

Electricity may also be employed. The following methods have been resorted to: *a.* Galvanization, either transversely through the mastoid processes, or longitudinally through the skull; or galvanization of the cervical sympathetic, the anode in the neck, the cathode below and behind the angle of the lower jaw. *b.* Faradization of the paralyzed muscles. *c.* Production of reflex movements of deglutition by the agency of the galvanic current, the anode being placed on the back of the neck, the cathode stroked rapidly and at short intervals over the sides of the larynx (feeble currents).

Special symptoms must sometimes be treated. Thus, Kayser treated salivation by subcutaneous injections of atropine (gr. $\frac{1}{6}$: 3 iiss., $\frac{1}{4}-\frac{1}{2}$ syringeful) in the region of the cheek, and Fauvel relieved dangerous attacks of suffocation by means of tracheotomy.

2. *Hemorrhage into the Medulla Oblongata.*

1. Very little is known concerning hemorrhages into the medulla oblongata, on account of their extreme rarity. They are associated somewhat more frequently with hemorrhages into the pons. Under such circumstances the symptoms may be identical with those of pons hemorrhage.

2. The causes are the same as in cerebral hemorrhage. The chief factor is the rupture of so-called miliary aneurisms; their rupture is favored by permanent increase of blood pressure (left ventricular hypertrophy) or its temporary increase (excitement, drinking). Secondary hemorrhage may occur in inflammation, softening, or compression of the medulla oblongata. Westphal produced it in guinea pigs by slight blows on the head.

The hemorrhage may be extremely minute (capillary), or it may be so extensive that the medulla oblongata is almost completely destroyed.

3. Hemorrhages of a certain extent give rise to sudden or very rapid death. The patients, with a cry, fall lifeless to the floor, or they become unconscious; epileptiform spasms develop not infrequently, the breathing becomes stertorous or assumes the Cheyne-Stokes type, the pulse is accelerated and irregular, hyperpyretic temperature sets in, and death occurs in a few minutes or hours.

Hemorrhages of smaller dimensions produce manifold symptoms. They generally begin like a stroke of apoplexy, the patients suddenly lose consciousness and do not regain it until after a certain length of time. Epileptiform convulsions are not infrequent, but appear to be due to irritation of the adjacent pons. Paralysis is produced in the limbs, but particularly in the bulbar nerves (acute or apoplectiform bulbar paralysis). But, on the one hand, acute bulbar paralysis may be entirely absent and, on the other hand, it may occur in thrombotic or embolic softening, acute inflammation, or compression of the medulla oblongata.

The motor paralysis not infrequently affects all the limbs. In other cases, it is hemiplegic in its distribution or, if the hemorrhage is very small, produces only monoplegia. Hemorrhage at the decussation may give rise to crossed paralysis, *i. e.*, paralysis of the arm on one side, of the leg on the opposite side of the body.

The paralyzed limbs present sensory disturbances if sensory fibres are also affected by the hemorrhage.

Paralysis of the bulbar nerves is associated with paralysis of the limbs if the hemorrhage affects the nuclei on the floor of the fourth ventricle, the nerve bundles in their intramedullary course, or the bulbar nerves at their points of exit. The facial, hypoglossus, glosso-pharyngeal, vagus, spinal accessory, acoustic, abducens, and trigeminus may be affected in varying combinations, unilaterally or bilaterally. The corresponding clinical symptoms are : paralysis of the face, tongue, palate, paralytic disturbance of articulation (anarthria), interference with

mastication and deglutition, dyspnœa, acceleration and irregularity of the pulse, singultus, vomiting, deafness, strabismus, and sensory disturbances in the face.

The hemorrhage may be situated in such a locality as to cause hemiplegia alternans, i. e., paralysis of the limbs on the side opposite to the hemorrhage, paralysis of the bulbar nerves on the same side. The hemorrhage has evidently affected the bulbar nerves after their (more central) decussation, while the pyramid tracts are affected before their decussation.

In some cases, the cerebral nerves are not paralyzed, but manifest evidences of irritation, particularly in the trigeminal distribution (trismus).

4. The diagnosis is only possible to one who possesses a thorough knowledge of the anatomy and physiology of the medulla. Even then, in the majority of cases, the intactness of the pons Varolii cannot be ascertained with certainty, nor can embolism or thrombosis of the arteries of the medulla be positively excluded.

The prognosis is very grave; the treatment is similar to that of cerebral hemorrhage.

3. *Embolism and Thrombosis of the Arteries of the Medulla Oblongata.*

1. The etiology and anatomical changes are the same as in cerebral embolism and thrombosis. Thrombosis is the result of compression, and of arterio-sclerotic and endarteritic changes (the latter often depend on syphilis); embolism usually follows endocardial changes in the left heart.

Thrombosis or embolism is followed by necrotic softening (red, yellow, or white) in the district in which the circulation of blood has been abolished. In some cases, only the finest arteries are affected, and fine, almost punctate infarctions are then found, particularly on the floor of the fourth ventricle. They are distinctly wedge-shaped, the base being directed upwards, the apex downwards.

Thrombotic and embolic softening of the medulla is sometimes associated with a similar condition in other parts of the brain.

2. Embolism and thrombosis in the medulla oblongata give rise to acute or apoplectiform bulbar paralysis. The symptoms vary, however, according to the vascular district in which the circulation has been abolished.

The medulla oblongata is supplied with blood by the vertebral and basilar arteries. The vertebral arteries supply direct vessels to the nerve roots alone, while the substance of the medulla is supplied by two main branches of the vertebral arteries, viz., the anterior spinal artery and the posterior inferior cerebellar arteries (vide Fig. 96, 12, *cbip*, 15, *spp*).

The branches of the anterior spinal artery pass at a right angle to the median fissure of the medulla, and their terminal arteries pass to the floor of the fourth ventricle, where they surround with a capillary network the nuclei of the hypoglossus, spinal accessory, and the supposed lower facial nucleus.

The inferior cerebellar artery supplies the pyramids, olivary bodies, and choroid plexus of the fourth ventricle, either directly or by means of the posterior spinal artery which springs from it.

Hence, the symptomatology will vary according as the thrombosis or embolism affects the anterior spinal artery or the posterior inferior cerebellar artery; the former is followed by paralysis of the hypoglossus, spinal accessory, and facial nerve, the latter by motor disturbances in the limbs. If the entire trunk of one of the vertebral arteries is occluded, the symptomatology will consist of paralysis of the bulbar nerves and the limbs. The left vertebral artery is plugged more frequently than the right, because it leaves the subclavian artery on the cardiac side of the arch, so that the current of blood runs more nearly in a straight line from the right subclavian.

The anterior spinal artery is not infrequently present on one side alone, and then is generally derived from the left vertebral. In such cases, the occlusion of one vertebral artery may possibly give rise to the symptoms of bilateral bulbar paralysis.

The basilar artery, by means of branches from its lower half, supplies the nuclei of the pneumogastric, glosso-pharyngeus, and acoustic, while the upper half sends branches to the nuclei of the facial (upper nucleus), abducens, oculomotor, and trochlearis. Extensive bulbar paralysis is to be particularly looked for when the vertebral and basilar arteries are both affected. Plugging of the lower part of the basilar artery is especially dangerous, since vagus paralysis may give rise to sudden death.

It is true that these considerations are schematic, but their proper understanding is necessary in order not to lose the thread in the labyrinth of innumerable variations which may be presented in different cases.

3. Sudden death may occur in thrombosis or embolism of the medulla oblongata, as in extensive hemorrhages into this locality.

In other cases, the symptoms are those of an attack of apoplexy, with loss of consciousness and occasionally with convulsions. The apoplectic onset may be absent if small vessels alone are affected or the thrombus forms slowly. Some patients suffer from dizziness and vomiting, or they complain of increasing weakness which, in a few hours, advances to complete paralysis.

The paralysis may be confined to the limbs (distribution of the inferior cerebellar artery) or to the bulbar nerves (anterior spinal and basilar arteries), or it affects both. It is sometimes unilateral, sometimes alternating, sometimes bilateral. Bilateral paralysis is sometimes the result of a unilateral lesion.

Fig. 96.
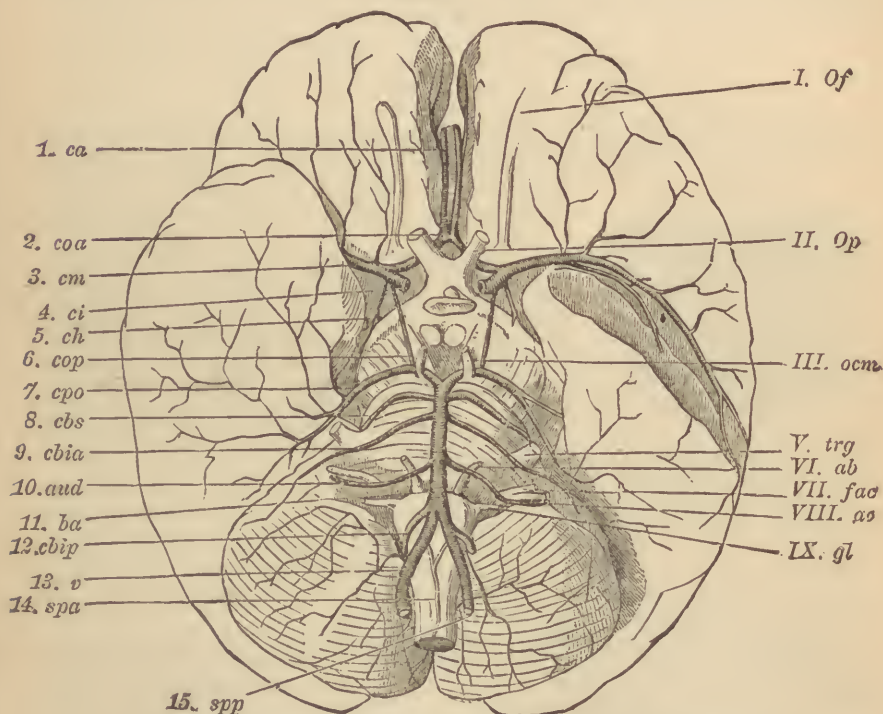
Distribution of the arteries at the base of the brain. After Henle.

1. *ca*, anterior cerebral artery; 2. *coa*, anterior communicating artery; 3. *cm*, middle cerebral artery; 4. *ci*, internal carotid artery; 5. *ch*, choroid artery; 6. *cop*, posterior communicating artery; 7. *cpo*, posterior cerebral artery; 8. *cbs*, superior cerebellar artery; 9. *cbia*, anterior inferior cerebellar artery; 10. *aud*, auditory artery; 11. *ba*, basilar artery; 12. *cbip*, posterior inferior cerebellar artery; 13. *v*, right vertebral artery; 14. *spa*, anterior spinal artery; 15. *spp*, posterior spinal artery; I. *Of*, olfactory nerve; II. *Op*, optic nerve; III. *ocm*, oculo-motor nerve; V. *trg*, trigeminus nerve; VI. *ab*, abducens nerve; VII. *fac*, facial nerve; VIII. *ac*, acoustic nerve; IX. *gl*, glosso-pharyngeus nerve.

The paralysis of the bulbar nerves produces effects similar to those described in the article on hemorrhage into the medulla. Sensory disturbances may be present or absent. Senator reported a case of thrombosis of the left vertebral artery with softening in the external, posterior, inferior portion of the left half of the medulla in which there was alternate paralysis of sensation, *i. e.*, anæsthesia of the left side of the face and the right limbs.

The paralyses may disappear gradually if the circulatory disturbances subside.

In other cases considerable improvement occurs. Or secondary degeneration develops in the pyramid tracts, and is shown by contractures and increase of the tendon reflexes in the affected extremities. The disease may then resemble amyotrophic lateral sclerosis, but is distinguished from it by the sudden onset and the absence of marked atrophy.

4. The differential diagnosis from hemorrhage is not always possible. Marked improvement of the paralysis, the existence of aortic or mitral lesions, previous syphilis, advanced age, and arterio-sclerosis of peripheral arteries favor the diagnosis of embolism and thrombosis.

Griesinger states that if both vertebrals or the basilar artery are occluded, compression of both carotids will produce epileptiform convulsions as the result of cerebral anæmia, but Leyden called attention to the danger associated with this experiment and to the fact that compression of the carotids may produce convulsions although the basilar is not occluded.

5. The prognosis is grave. The treatment is the same as that of cerebral embolism and thrombosis. If there is paralysis of deglutition, food must be introduced through the œsophageal sound.

4. Acute Inflammation of the Medulla Oblongata. (Bulbar Myelitis.)

1. Three cases observed by Leyden prove that acute inflammatory processes may develop in the medulla oblongata as in the spinal cord. We will not now consider the secondary inflammations following thrombosis, embolism, hemorrhage, and compression of the medulla, or which extend to this organ from the spinal cord.

Fig. 97.

Transverse section through the medulla oblongata, with numerous hemorrhagic inflammatory foci. After Leyden. Enlarged 1½ times. The individual foci are darkly shaded.

2. The disease occurred in irregularly scattered, usually small foci which were occasionally visible only after microscopical examination. These foci were in a condition of red softening. The walls of the vessels were thickened, their nuclei increased, the lymphatic sheaths were filled with cellular elements; there were perivascular hemorrhages, granulo-fatty cells, and swelling of the axis-cylinders.

3. The causes of the disease are unknown. One of Leyden's cases occurred in a drunkard, the two others had suffered from articular rheumatism.

4. The symptoms are those of an acute, apoplectiform bulbar paralysis, with or without paralysis of the limbs. Different nerves are affected according to the number, situation, and size of the inflammatory foci. There is slight elevation of temperature. The disease always terminates fatally, with increasing difficulty of respiration. Duration of the disease, four to ten days.

5. The differentiation of the disease from hemorrhage, embolism, softening, or compression is facilitated by the following features: the absence of an apoplectic onset and convulsions, the presence of slight fever, and increase of the symptoms until the fatal termination.

6. The disease is always fatal. The treatment is the same as that of acute myelitis (vide page 88).

5. Tumors of the Medulla Oblongata.

1. Tumors which develop in the medulla and are confined to this part are rare. In 1881, Bernhardt had collected eighteen cases, and very few have been observed since.

2. Solitary tubercles constitute the most frequent variety. One to three are found, in the shape of yellow, cheesy nodules, which may attain the size of a walnut. Next in frequency are the gliomata. R. Schultz reports a case in which a glioma was found at the bottom of a cyst filled with brown fluid (previous softening?). Mosler and Virchow have described a gliosarcoma. Fibroma, myxoma, papilloma, and carcinoma are also mentioned. The possibility of the development of gummata must also be taken into consideration. The tumors may attain the size of a pigeon's egg.

3. We must distinguish between general and local symptoms. The former merely indicate the presence of a compressing object within the skull, the latter are manifested by irritative or paralytic phenomena on the part of the bulbar centres and nerves.

The general symptoms include headache (particularly in the back of the neck and occiput, and often increasing into paroxysms), frequent vomiting, dizziness, staggering gait, choked disc, impairment of vision, and epileptiform convulsions.

The chief local symptoms are irritation and paralysis of the bulbar nerves and nuclei (strabismus, paralysis of the face, tongue, pharynx, palate, vocal cords, tinnitus aurium, and impairment of hearing, respiratory disturbances, and slowness of the pulse and obstinate singultus. Paralysis of the limbs and sensory disturbances may also arise; ataxic movements have been described. Polyuria and mellituria are indicative of implication of the fourth ventricle. Quioc observed forced deviation of the head and eyes to the right in a case of tumor of the left half of the medulla.

Among eighteen cases Bernhardt found:
Headache, in more than half the cases.
Vomiting, in more than half the cases.
Dizziness, six times.
Epileptiform convulsions, one time.
Diplopia, three times.
Impairment of sight, five times.
Dilatation of the pupils, five times.
Paralysis of ocular muscles, in more than half the cases.
Auditory disturbances, four times.
Speech disturbances, four times.
Difficulty in deglutition, one time.
Singultus, two times.
Retardation of the pulse, two times.
Difficulty in breathing, two times.
Changes in the urinary secretion, three times.

In some cases, however, the tumors give rise to no symptoms. They are sometimes found accidentally, or may give rise to sudden death. Schultz recently reported a case which had presented all the symptoms of spastic spinal paralysis.

The mental condition is often affected; in almost half the cases reported by Bernhardt, mention is made of impairment of memory, confusion of ideas, dulness, and somnolence.

4. The diagnosis presents numerous difficulties and is often entirely impossible. As a rule, it can be made only with a certain degree of probability. The diagnosis is easiest when the general and local symptoms are present in their completeness. It is distinguished from progressive bulbar paralysis by the fact that no general symptoms are noticeable in the latter disease.

5. The prognosis is relatively favorable in syphilis; otherwise it is unfavorable. The treatment is purely symptomatic.

6. *Injuries, Acute or Slow Compression of the Medulla Oblongata.*

1. Injuries to the medulla are produced by stab and gunshot wounds and blows. As a rule, death takes place at once. The patients fall lifeless to the ground, with or without a cry and epileptiform convulsions.

2. Injuries sometimes give rise to the symptoms of acute compression. This is observed in fractures and dislocations of the upper cervical vertebræ, particularly the atlas and axis (as the result of a fall or blow, or of tuberculous processes). Dislocation of the odontoid process occurs with relative frequency, and the bone then presses backwards on the medulla. It is a noteworthy fact that tubercular processes may develop in the vertebræ in a latent manner, and give rise unexpectedly to a fatal termination. Sudden hemorrhage, for example, from a ruptured aneurism of the basilar artery, may produce rapid death, with symptoms of acute compression of the medulla. If the injury is slight, the maintenance of life is conceivable; the acute development of bulbar symptoms would indicate a lesion of the medulla.

3. Slow compression of the medulla often depends upon thickenings or accumulations of pus at the occiput or upper cervical vertebræ, such as occur not infrequently in tuberculosis. Thickening of the odontoid process (sometimes, perhaps, the result of rachitis) occasionally gives rise to compression; or this may be produced by an abnormal condition of the occiput or posterior arch of the

atlas and odontoid process. The compression is occasionally the result of deforming arthritis of the joints between the occiput, atlas, and odontoid process, particularly if the latter has undergone thickening. The same mechanical effects will be produced by tumors growing from adjacent parts, and by aneurisms.

Compression of the medulla not alone exercises a mechanical effect, but also gives rise to a compression myelitis.

The clinical history consists of bulbar irritative and paralytic phenomena, the former appearing at the onset, the latter at the close. The symptomatology varies according to the site of compression. Paralysis sometimes appears in the limbs earlier than in the bulbar nerves. Spastic spinal paralysis has been observed when the pyramid tracts were chiefly affected. The diagnosis may be impossible. Prognosis usually unfavorable. Treatment symptomatic.

PART IV.

DISEASES OF THE BRAIN.

Diagnostic Preliminary Remarks.

Since the time of Griesinger, we distinguish between diffuse cerebral symptoms and focal symptoms in diseases within the cranial cavity.

The diffuse cerebral symptoms are of a general nature, and merely indicate that the brain has been affected in some way. They are the result of changes of pressure within the cranium, of circulatory disturbances, of so-called general shock, or of a number of factors. These symptoms include: vertigo, clouded consciousness, impairment of intelligence, flashes of light before the eyes, ringing in the ears, vomiting, changes in the frequency and rhythm of the pulse, irregularity in respiration, epileptiform convulsions, etc. Certain diseases within the skull produce diffuse symptoms alone, for example, affections of the meninges.

The focal symptoms, on the other hand, possess a special diagnostic significance because they indicate a definite site of disease in the brain. They are divided into two groups, viz.: irritative and paralytic symptoms. Like the diffuse symptoms, the focal symptoms may exist as such from the start, but as a general thing they may be looked for only when the lesion develops slowly, is small in extent, and does not possess those properties which would give rise to the development of diffuse cerebral symptoms.

Diffuse and local symptoms are often associated with one another. The former are often present at the start, but the more they subside the more distinct the focal symptoms become. This is well illustrated in cerebral hemorrhage. On the other hand, focal symptoms may open the scene, and are followed by diffuse symptoms, the more the lesion increases in extent.

The focal symptoms may be direct or indirect, temporary or permanent.

The indirect focal symptoms are the result of a sort of action at a distance. The lesion is perhaps remote from the part towards which the symptoms point, but it acts upon the latter from a distance in such a manner as if the part in question were directly injured. Almost all cerebral lesions possess the peculiarity of exercising a more or less distinct remote action, and of impairing the cerebral functions beyond their proper boundaries. In many of them the remote effects disappear after a time (temporary focal symptoms). The symptoms which remain perma-

nently, because the lesion has produced irreparable changes in the parenchyma of the brain, constitutes the permanent focal symptoms.

A distinct line cannot always be drawn between focal and diffuse symptoms, or between the different varieties of focal symptoms. Vomiting, for example, is often a diffuse symptom, but sometimes attains the dignity of a focal symptom.

a. Focal Symptoms in Diseases of the Cerebral Cortex.

The motor functions of the brain are confined almost exclusively to the anterior and posterior central convolutions and the paracentral lobule.

FIG. 98.
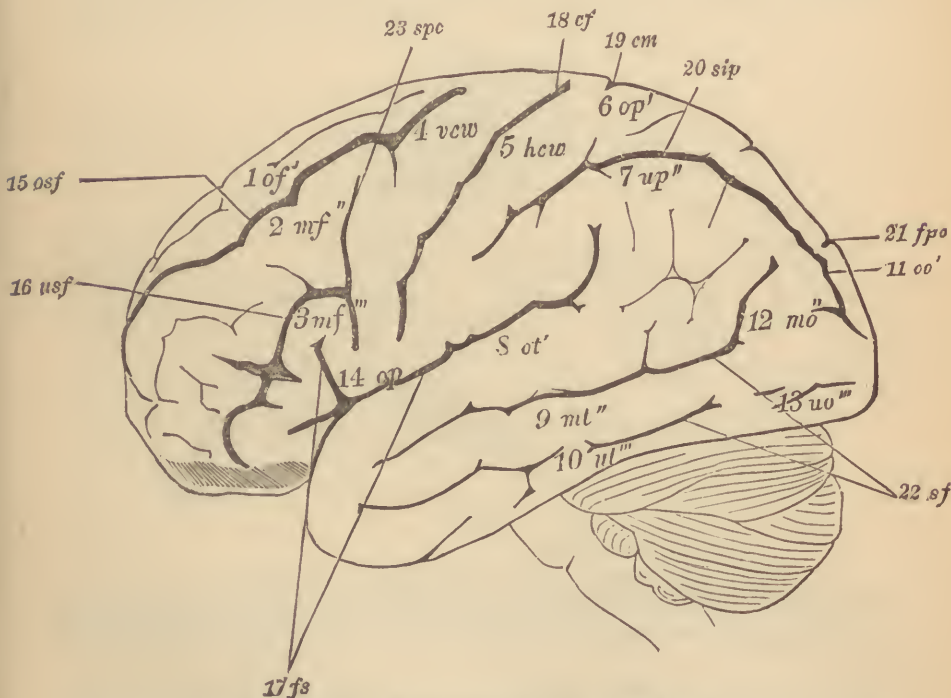
Convolutions of the surface of the brain. After Ecker.
1 *of'*, superior (first) frontal convolution; 2 *mf''*, middle (second) frontal convolution: 3 *mf'''*, inferior (third) frontal convolution; 4 *vcw*, anterior central convolution; 5 *hcw*, posterior central convolution; 6 *op'*, superior parietal lobule; 7 *up''*, inferior parietal lobule: 8 *ot'*, superior (first) temporal convolution; 9 *mt''*, middle (second) temporal convolution; 10 *ut'''*, inferior (third) temporal convolution; 11 *oo'*, superior (first) occipital convolution; 12 *mo''*, middle (second) occipital convolution; 13 *uo'''*, inferior (third) occipital convolution; 14 *op*, operculum; 15 *osf*, superior frontal fissure; 16 *usf*, inferior frontal fissure; 17 *fs*, fissure of Sylvius, horizontal ramus on the right side, ascending ramus on the left side; 18 *cf*, fissure of Rolando; 19 *cm*, calloso-marginal fissure; 20 *sip*, interparietal fissure; 21 *fpo*, parieto-occipital fissure; 22 *sf*, superior (first) and inferior (second) temporal fissures; 23 *spc*, præcentral fissure.

This region is easily found on the surface of the brain. The chief landmark is the fissure of Rolando, which begins near the median border of the hemisphere and runs obliquely forwards and downwards (Fig. 98, 18 *cf*). Its lower extremity is situated between the two rami of the Sylvian fissure, of which the posterior is known as the horizontal ramus, the anterior as the ascending ramus. Anteriorly the fissure of Rolando is bounded by the anterior central convolution

(Fig. 98, 4 *vcw*), posteriorly by the posterior central convolution (Fig. 98, 5 *hcw*), both merge into one another above and below the fissure of Rolando. The paracentral lobule is visible on the median aspect of the hemisphere (Fig. 99, 1 *lprc*), and is the direct continuation of the central convolutions. It is bound posteriorly by the calloso-marginal fissure which separates it from the præcuneus (Fig. 99, 5 *pc*), and its anterior boundary is formed by a prolongation of the calloso-marginal fissure (Fig. 99, 4 *scm*), to which Schwalbe applies the term paracentral fissure (Fig. 99, 2 *sprc*).

The symptoms of an affection of the motor regions of the cortex will vary according as the brain substance is destroyed or irritated. In the former event paralysis, in the latter spasm of the muscles will be produced. But both symptoms are often associated with one another, and this very feature is, to a certain extent, pathognomonic of cortical lesions.

The distribution of the paralytic and spasmodic phenomena depends

FIG. 99.
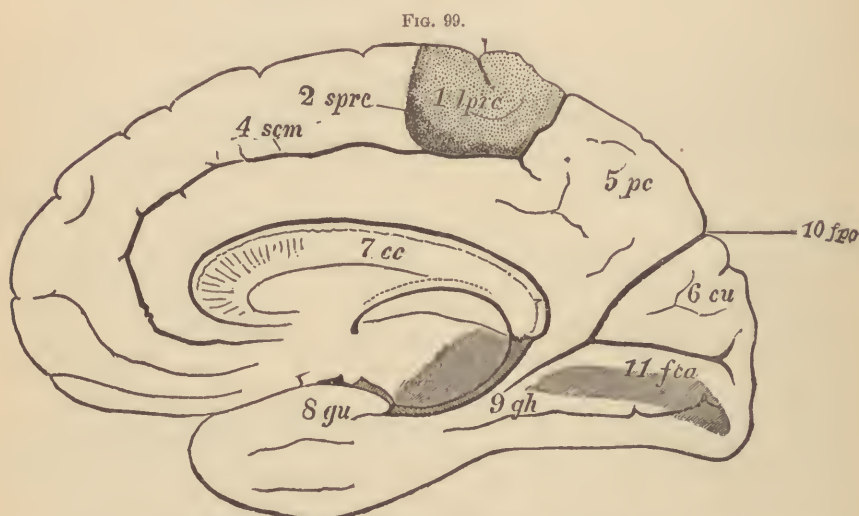
Median aspect of the surface of the brain.. After Ecker.
1 *lprc*, paracentral lobule (leg centre) ; 2 *sprc*, paracentral fissure ; 3 *sc*, end of the fissure of Rolando ; 4 *scm*, calloso-marginal fissure ; 5 *pc*, præcuneus ; 6 *cu*, cuneus ; 7 *cc*, corpus callosum ; 8 *gu*, gyrus uncinatus ; 9 *gh*, gyrus hippocampi ; 10 *fpo*, parieto-occipital fissure ; 11 *fca*, calcarine fissure.

upon the extent of the lesion. It is characteristic of circumscribed cortical affections that they not infrequently produce monoplegias of the face, arm, leg, or even of small nerve tracts, with the character of a central paralysis in regard to the electrical current (no degeneration reaction).

If the entire motor region of the cortex is destroyed, the symptoms of ordinary cerebral hemiplegia are produced, *i. e.*, the arm and leg are paralyzed on the side opposite to the lesion, and also the facial nerve so far as regards the branches to the mouth and cheeks, while the frontal branch is unaffected, and the patient retains the power of wrinkling the forehead and closing the lid. In uncomplicated cases, sensory and vasomotor disturbances are absent, but ptosis (paralysis of the oculo-motor nerve) has been observed in some cases. The paralysis is exactly like that produced by destruction of the internal capsule, or certain portions

of the centrum ovale which contain the radiating fibres from the internal capsule.   At times the cortical origin of the paralysis can be inferred from the etiology alone.   This includes particularly injuries to the skull and embolism or thrombosis of cortical arteries.   The two latter processes generally involve the middle cerebral artery, which supplies the motor cortical region.

But as this artery also supplies the third frontal convolution and island of Reïl, these parts are often affected in addition to the motor region proper, and aphasia will result if the lesion is situated on the left side.   In diffuse cortical paralysis, as in that produced by lesion of the internal capsule, improvement is generally more marked in the leg than in the arm; after a while contractures, spasms, and increase of the tendon reflexes make their appearance, the result of secondary degeneration of the pyramid tracts from the cerebral cortex to the spinal cord.

FIG. 100.

Schematic representation of the position of the motor centres of the cortex of man.
(Leg centre in the paracentral lobule, vide Fig. 99.)

The more circumscribed the cortical lesion, the more the paralysis will be confined to the distribution of individual nerves (nerves of the arm or leg, facial, hypoglossal, and motor oculi nerves).

So far as known at present, the motor cortical centres are located as follows:

a. Centre for the leg in the upper third of the anterior central convolution and the paracentral lobule, and in the upper two-thirds of the posterior central convolution.

b. Centre for the arms in the middle third of the anterior (perhaps also the posterior) central convolution.

c. Centre for the facial nerve in the lower third of the anterior central convolution.

d. Centre for the hypoglossal nerve, in the lower part of the anterior central convolution (vide Fig. 100).

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Hence, if the lesion extends in circumference, the paralysis may spread from one motor tract to an adjacent one. For example, if the lesion began in the arm centre and spread upwards, it will be followed by paralysis of the leg; if it spread downwards, by paralysis of the facial nerve. Cortical paralysis of the leg and face alone is hardly conceivable, since the lesion must then have jumped over the motor tract of the arm. The gradual conversion of a monoplegia into an incomplete and finally, perhaps, a complete hemiplegia is characteristic of cortical disease.

Disease of the motor part of the cortex may also produce muscular spasms and epileptiform convulsions, whose distribution varies according to the extent of the irritating lesion. But an unilateral cortical lesion may also give rise to general convulsions. Consciousness may or may not be retained during the attacks. In the latter event they may be mistaken for ordinary epilepsy, but in cortical (Jacksonian) epilepsy the convulsions always began in a certain limb, or are confined to that part.

The not infrequent combination of paralysis and epileptiform twitchings is especially characteristic of disease of the cortex. As a general thing, the twitchings follow the paralysis.

The following data favor the diagnosis of an affection of the motor cortical centres:

a. Monoplegias of a central quality as regards the electrical current (no degeneration reaction).

b. Hemiplegias with implication of the motor oculi nerve (ptosis), later contracture and increased tendon reflexes as the result of secondary degeneration of the pyramid tracts.

c. Development of hemiplegias from the combination of circumscribed paralyses ; owing to the gradual spread of the lesion to adjacent cortical motor centres.

d. Epileptiform convulsions in individual nerve tracts, or general epileptiform convulsions, which always begin in the same limb, and are often attended with loss of consciousness (Jacksonian or cortical epilepsy).

e. A combination of spasms and paralysis in individual nerve tracts, or in a hemiplegic form.

We will now consider the lesions of other portions of the cortex.

Diseases of the cortex of the frontal lobe are related to certain disturbances of speech, known as aphasia, which will be discussed in detail at a later period. It will here suffice to remark that motor or ataxic aphasia is associated with lesions of the pars opercularis of the third frontal convolutions (Fig. 98, 3 mf'''). The larger part of the frontal cortex is probably associated with purely psychical processes; it has been found to be markedly atrophied in dementia paralytica and idiocy.

Diseases of the cortex of the temporal lobe are related, under certain circumstances, to auditory disturbances and, as a consequence, to sensory aphasia (word-deafness). According to Wernicke, this form of aphasia occurs only when the lesion is situated in the first (superior) temporal convolution. The occurrence of deafness in such cases has not been positively proven. For further details, vide page 205.

Destruction of the cortex of the parietal lobe, in which, according

to Flechsig, the sensory tracts of the skin terminate, is said to produce loss of muscular sense. The patients are no longer able, when the eyes are closed, to tell the position of the limbs. This symptom may be associated with paralysis, if the motor portions of the cortex are also implicated.

Lesion of the cortex of the occipital lobe is associated with visual disturbances (hemianopsia or hemiopia).

In order to understand this symptom, we must bear in mind that a partial decussation of the optic fibres takes place in the optic chiasm, and that in both optic nerves the lateral portions remain upon the same side, while the median portions decussate (vide Fig. 101). Hence the

FIG. 101.
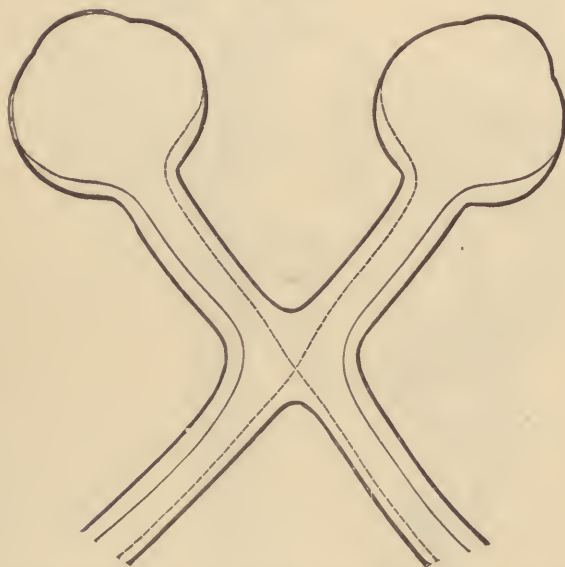
Schematic representation of the decussation of the optic nerve fibres in the chiasm.

left optic nerve, for example, supplies not alone the temporal side of the left eye, but also the nasal portion of the right eye. If we now assume that each optic nerve terminates centrally in the occipital cortex of the same side, it follows that lesions of the left occipital lobe will give rise to an affection of both eyes, the left eye becoming blind upon the temporal half, the right eye upon the nasal half of the retina. In other words, the right half of the field of vision in each eye is lost, i. e., there is right hemianopsia. Lesions of the right occipital lobe will produce left hemianopsia.

In some cases the hemianopsia is not complete, and affects in unequal degrees the senses of light, color, and space.

Fuerstner noticed that insane patients, who suffered from diseases of the occipital lobe, were able to see objects, but did not recognize them, so that the optical images appeared to have been lost (psychical blindness).

b. *Focal Symptoms in Diseases of the Centrum Ovale.*

The centrum semiovale is the mass of white fibres which is situated between the cerebral cortex and the basal ganglia. It is formed of fibres which differ very much in function. Some connect different parts of the cortex of the same hemisphere (association system). The symptoms produced by their destruction are unknown. Other fibres belong to the system of the corpus callosum, *i. e.*, through the medium of the latter they connect parts of the cortex of one hemisphere with identical parts in the other hemisphere (commissural system). Concerning the effects of their destruction we are also in the dark. Of special importance is the radiation of the

Fig. 102.
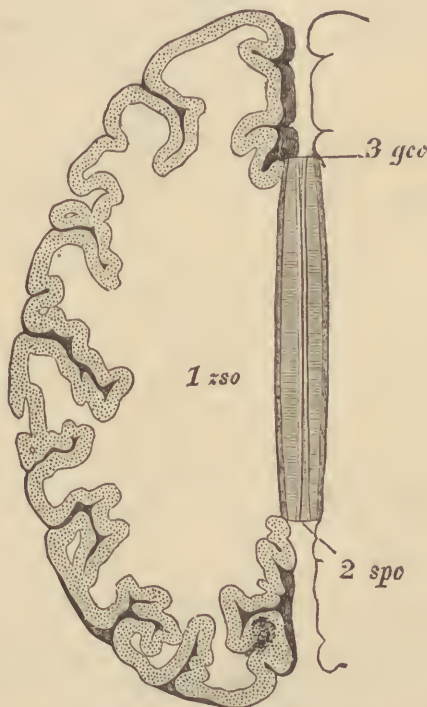
Horizontal section at the level of the surface of the corpus callosum.
1 *zso*, centrum semiovale ; 2 *spc*, splenium corporis callosi ; 3 *gcc*, genu corporis callosi.

middle portion of the crus cerebri, which first enters the internal capsule and then passes into the cortex (Fig. 103) (corona radiata). Since this part is the means of conduction between the cerebral cortex and the periphery of the body, interruption of conduction will produce, as a matter of course, the same symptoms as a lesion of the cortex itself, and during life we are unable to distinguish between the two processes. The distinction is often difficult anatomically, and we cannot always decide with certainty whether the lesion is situated in the cortex alone, or also implicates the immediately adjacent portion of the corona radiata. In diseases of the corona radiata, the symptoms vary according to the bundles of fibres which are affected. If those fibres which pass to the

central convolutions are affected, small, circumscribed lesions will produce monoplegia of this or that part; extensive lesions give rise to hemiplegia of the other half of the body. Destruction of the fibres passing

FIG. 103.
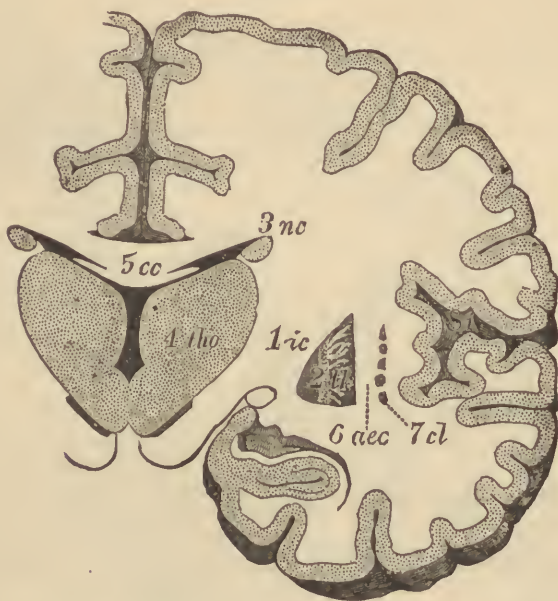
Frontal section through the cerebrum. 1 *ic*, internal capsule ; 2 *lk*, lenticular nucleus with its three divisions, external, middle, and internal ; 3 *nc*, caudate nucleus ; 4 *tho*, optic thalamus ; 5 *cc*, corpus callosum ; 6 *aec*, external capsule ; 7 *cl*, claustrum ; 8 *i*, island of Reil.

to the upper temporal lobe produces word-deafness, to the occipital lobe hemianopsia, etc.

### c. *Focal Symptoms in Diseases of the Internal Capsule.*

The internal capsule is that mass of fibres which is bounded internally by the optic thalamus and caudate nucleus, externally by the lenticular nucleus (vide Figs. 103 and 104). It presents an anterior and posterior division, the former situated between the caudate nucleus and lenticular nucleus, the latter between the optic thalamus and lenticular nucleus. Both divisions meet at the knee of the internal capsule (vide Fig. 104).

The internal capsule receives the motor and sensory fibres passing from the cortex to the crus cerebri. Hence its destruction is attended with motor and sensory paralysis.

The motor tracts are situated in the posterior division of the internal capsule, about at the posterior end of its middle third, the paths of conduction for the facial nerve being situated in front of those for the limbs. The sensory tracts for the skin, muscles, and cerebral nerves are situated in the posterior third of the posterior division (vide Fig. 104). The symptoms produced by lesions of other parts of the internal capsule are unknown.

A lesion of the middle third gives rise to the typical symptoms of cerebral hemiplegia, *i. e.*, to permanent paralysis in the upper and lower limbs, trunk, muscles, and facial nerve on the opposite side of the body, on account of the interruption to conduction to the motor portions of

FIG. 104.
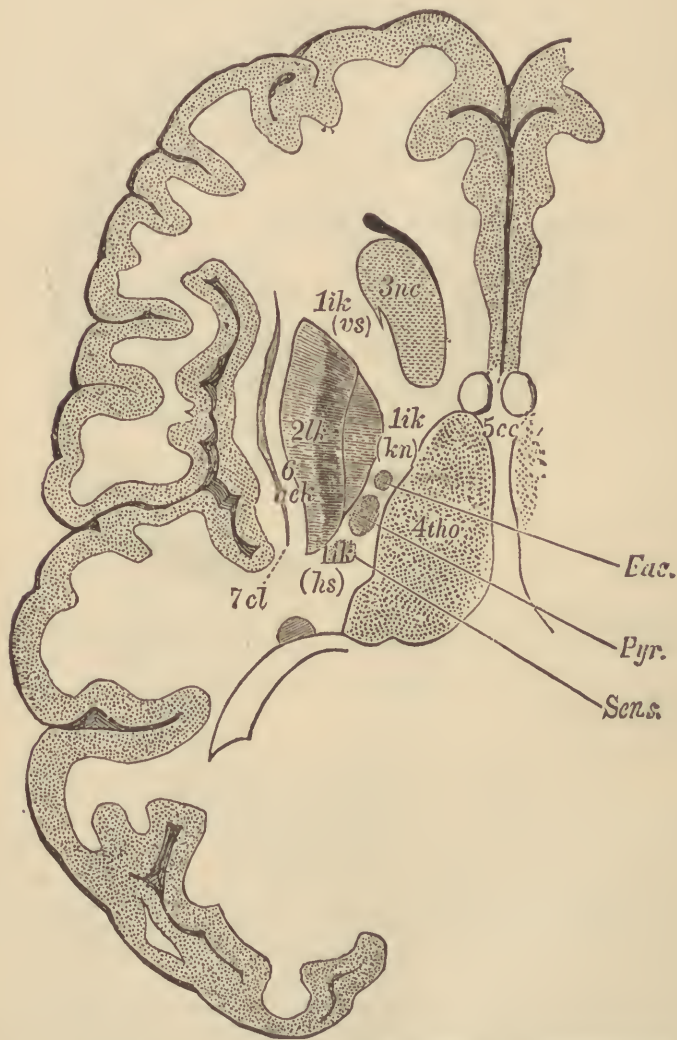
Horizontal section through the cerebrum. 1 *ik*, internal capsule ; *vs*, anterior division, *hs*, posterior division, *kn*, knee of internal capsule ; 2 *lk*, lenticular nucleus ; 3 *nc*, caudate nucleus ; 4 *tho*, optic thalamus ; 5 *cc*, corpora candicantia ; 6 *aek*, external capsule ; 7 *cl*, claustrum ; *Fac*, facial fibres in the posterior division of the internal capsule ; *Pyr*, pyramid fibres for the limbs ; *Sens*, Sensory tracts.

the cortex. The frontal branch of the facial nerve, which innervates the frontalis, corrugator supercilii, and orbicularis palpebrarum, remains unaffected, so that the patients are able to wrinkle the brow and close

the eyes. The hypoglossal nerve also is often paralyzed. If the paralysis has lasted for some time, secondary degeneration is often produced; it extends on the peripheral side of the lesion through the pyramid tract of the corresponding crus (middle third) and pons Varolii, then through the decussation of the pyramids into the lateral column-pyramid tract of the opposite side of the cord, and the anterior column-pyramid tract of the same side.

Lesions which are confined to the motor portion of the internal capsule are rather exceptional, and it is particularly noticeable in cerebral hemorrhages that the corpus striatum is also affected. This is owing to the fact that the distribution of cerebral lesions depends upon the distribution of the blood-vessels, and the internal capsule and corpus striatum are supplied by the same vessels. The usually simultaneous affection of the internal capsule and corpus striatum long led to the erroneous notion that cerebral hemiplegia depended on destruction of the latter. At the present time the motor tracts of the internal capsule are held responsible for the symptoms, and it is not known whether the lesion of the corpus striatum produces any change in the clinical history.

Lesions of the posterior third of the posterior division of the internal capsule are followed by cerebral hemianæsthesia. On the opposite half of the body the patients suffer from complete cutaneous anæsthesia, which terminates abruptly at the median line. The mucous membranes of the lips, mouth, nose, conjunctiva, external auditory meatus, penis, vagina, and rectum are also anæsthetic. The eye, ear, nose, and tongue are often, but not constantly, hemianæsthetic. The disease is often associated with vaso-motor disturbances, viz., redness of the skin, elevation of temperature, and diaphoresis.

Lesions which affect the middle and posterior thirds of the posterior division of the internal capsule are followed by hemiplegia and hemianæsthesia.

Some authors attribute præhemiplegic and posthemiplegic chorea and athetosis to disease of the hindmost portions of the posterior division of the internal capsule, but this theory has not been sufficiently well established.

### d. Focal Symptoms in Diseases of the Basal Ganglia.

The term basal ganglia is applied to the lenticular nucleus (Figs. 103 and 104, 2 *lk*) and caudate nucleus (Figs. 103 and 104, 3 *nc*). Lesions may be present in both ganglia, without giving rise to permanent motor disturbances. Transitory paralyses are observed more frequently, but are probably the result of pressure on the adjacent internal capsule.

e. Focal Symptoms in Diseases of the Crura Cerebri.

The characteristic symptom of diseases of the crus cerebri consists of alternate hemiplegia with regard to the motor oculi nerve, *i. e.*, the upper and lower limbs, generally the facial and hypoglossal nerves, even the trigeminus, are paralyzed upon the side of the body opposite to the lesion; the motor oculi is paralyzed on the same side. This striking feature is readily understood. Fig. 95 III. *ocm* shows the intimate relation of this nerve to the crus cerebri, and this is shown still more distinctly on a transverse section through the crus (vide Fig. 105). It contains two strata, the lower one or foot of the peduncle being more important clinically, because it contains in its middle portion the pyra-

mid tract, which passes from the central convolutions through the
internal capsule and the peduncle of the pons, medulla, and pyramid
tracts in the cord. The upper stratum of the crus is known as the teg-
mentum. Both strata are separated by a narrow zone of blackish-brown
substance (substantia nigra) which results from marked pigmentation of
the ganglion cells in this locality. Paralysis of the limbs is produced
by lesions of the crus only when the pyramid tract is injured. The
paralysis occurs almost always upon the side opposite to the lesion,
because the larger part of the pyramid tract decussates in the pyramids.
Paralysis of the limbs on the same side as the lesion is only conceivable
if there is no decussation. The root fibres of the motor oculi nerve pass
to the median portion of the substantia nigra, pass through it, and ex-
tend upwards to their nucleus (Fig. 105, 1 *ocm* and 2 *kocm*), which,
situated alongside the raphe, occupies the space between the pos-

<div align="center">Fig. 105.</div>
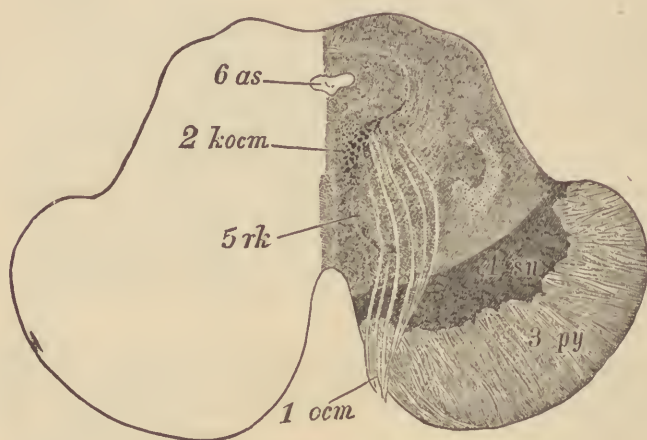
Transverse section of the cerebral peduncle, corresponding to the posterior part of the anterior
corpora quadrigemina. Enlarged 3 times. 1 *ocm*, oculo-motor nerve ; 2 *kocm*, nucleus of the
oculo-motor nerve ; 3 *py*, pyramid tract ; 4 *sn*, substantia nigra ; 5 *rk*, red nucleus ; 6 *as*, aque-
duct of Sylvius.

terior commissure and the border of the anterior and posterior corpora
quadrigemina. Hence it is evident that the paralysis of the motor oculi
nerve will not be crossed like that of the limbs, since the fibres of the
nerve decussate on the central aspect of the nucleus, and, therefore, of
a lesion of the crus. Moreover, this nerve may not be paralyzed in
lesions of the crus, if the median portion of the crus remains unaffected
or if the lesion is situated, not in the neighborhood of the pons, *i. e.*,
near the point of entrance of the nerve, but in the anterior half of the
crus. Under the latter circumstances an hemiplegia is produced which
cannot be distinguished from that occurring in lesions of the internal
capsule. Lesions of very small extent, if they are situated in the ex-
ternal portion of the peduncle, may be lacking in symptoms, since the
pyramid tract is situated in the middle portion (Fig. 71).

Paralysis of the motor oculi nerve is easily recognized. On account

. of paralysis of the levator palpebræ superioris, the upper lid droops upon the paralyzed side (ptosis) and cannot be drawn up voluntarily; internal rotation of the eye is impossible on account of paralysis of the internal rectus, and the eye is drawn permanently outwards on account of the preponderance of the external rectus which is applied by the abducens. Movements of the eye upwards and downwards are also abolished and impaired on account of paralysis of the superior and inferior recti and inferior oblique muscles. The patients complain of double vision, the pupil is dilated (mydriasis), and does not react to light.

Hemiplegia with alternate paralysis of the motor oculi may also occur if an ordinary hemiplegia is associated with a basal process which paralyzes the nerve on the side of the lesion in the cerebrum, and a differential diagnosis is not always possible under such circumstances.

The tegmentum of the crus contains sensory tracts, which pass to the posterior third of the internal capsule, and thence to the cerebral cortex (vide Fig. 104, *Sens*). Although it is very probable that lesions of the tegmentum will give rise to sensory disturbances, nothing is known positively in this regard.

f. Focal Symptoms in Diseases of the Pons Varolii.

Although slight lesions sometimes produce rapid death, morbid processes in this region may be entirely latent. This is sometimes true of

FIG. 106.
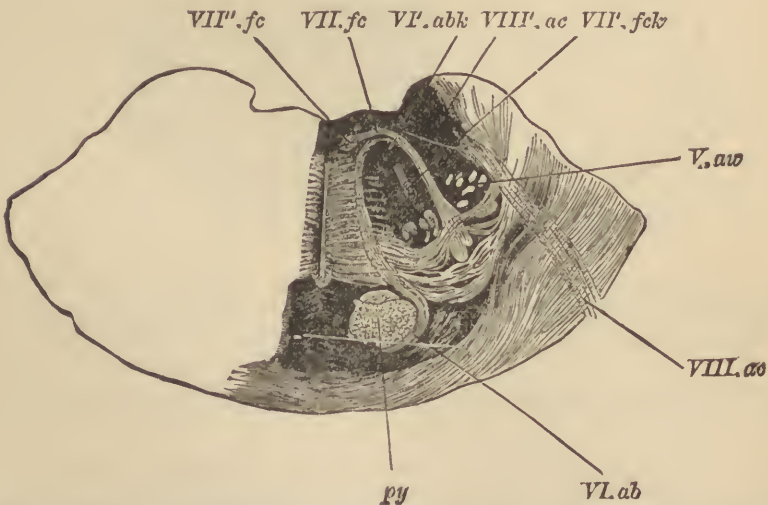
Transverse section of the pons Varolii about the middle of the fourth ventricle. Enlarged 3 times. V. *aw*, ascending trigeminus root ; VI. *ab*, abducens nerve ; VI'. *abk*, abducens nucleus ; VII. *fc*, facial nerve ; VII'. *fc*, facial nucleus ; VII''. *fc*, intermediate portion of facial on transverse section ; VIII. *ac*, anterior acoustic root ; VIII'. *ac*, nucleus of anterior acoustic root ; *py*, pyramid tract.

tubercles of the pons, probably because they slowly separate, but do not destroy the nerve fibres.

A characteristic feature of diseases of the pons is alternate hemi-

plegia, in which the facial nerve is paralyzed on one side of the body (the same side as the lesion), the limbs upon the opposite side. At the same time, all the branches of the facial nerve are paralyzed as in peripheral paralysis. Furthermore, degeneration reaction develops in the paralyzed facial muscles.

Other cerebral nerves (hypoglossus, abducens, trigeminus) on the same side may also be paralyzed.

Alternate hemiplegia is not, however, a necessary result of unilateral pons lesions. It will only occur if the lesion is situated in the medullary (lower) half of the pons, since it is the result of interruption to conduction in the facial nerve on the peripheral aspect of its decussation (which takes place in the upper part of the pons), while the fibres

Fig. 107.
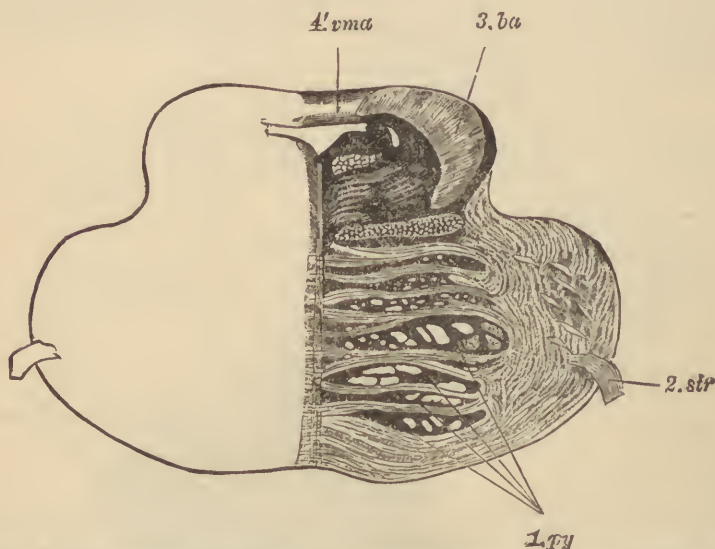
Transverse section through the pons near its peduncular extremity. Enlarged 3 times. 1 *py*, pyramid tract; 2 *str*, sensory trigeminus root; 3 *ba*, superior cerebellar peduncles; 4 *vma*, velum medullare anterius.

which pass to the limbs (pyramid tract) are interrupted on the central side of their decussation.

If the lesion is situated in the upper (peduncular) portion of the pons, the facial nerve and the pyramid tract are injured above their decussation, so that the face and limbs are paralyzed on the opposite side of the body. At the same time, the frontal branch of the facial nerve remains unaffected, as it does in cerebral hemiplegia, and degeneration reaction is not produced. Under such circumstances, the diagnosis may be very difficult unless other pons symptoms are present. The latter must be present in a certain combination in order to render the diagnosis certain. The pons symptoms include: *a*, paralysis of other cerebral nerves, especially the trigeminus, abducens, acoustic, hypoglossus, occasionally the vagus, spinal accessory, and glosso-pharyngeus (the two latter as the result of pressure on the medulla), and the motor oculi, if the peduncle is affected; *b*, disturbances in articulation (anarthria); *c*, disturbances of deglutition

(dysphagia); d, marked contraction of the pupils; e, a tendency to epileptiform convulsions. The motor paralysis of the limbs is associated not infrequently with anæsthesia; according to some writers, this only occurs in lesions of the lateral third of the pons (vide Fig. 106).

The diagnosis is especially difficult when individual systems of fibres are alone affected by the lesion. If the pyramid tracts are alone affected (Fig. 107, 1 py), the limbs will be paralyzed on the opposite side of the body if the lesion is unilateral, indeed monoplegia is occasionally produced. Lesions near the floor of the fourth ventricle will give rise to paralysis of the cerebral nerves in different combinations, according to the distribution of the lesion. The nerves may be paralyzed on the side of the lesion, or on the opposite side, according as they are injured above the decussation (paralysis on the opposite side) or below it (paralysis on the same side).

Lesions near the median line may give rise to paraplegic symptoms, either because the conduction is interrupted on both sides, or because the unilateral lesion produces pressure-effects upon the opposite side. If the compression disappears, the symptoms dependent on it will also subside. The symptomatology is extremely varied, because sometimes the limbs are paraplegic, while the cerebral nerves are paralyzed in a hemiplegic manner, or vice versa, or the cerebral nerves present a combination of hemiplegic and paraplegic symptoms. The problem can be solved only by those who possess a certain amount of anatomical knowledge, and we again refer the reader to Figs. 106 and 107.

APPENDIX.

The diagnosis of diseases of the optic thalamus is generally impossible. At all events, they are not associated with motor paralysis. Destruction of the posterior third of the thalamus gives rise to visual disturbances similar to those produced by lesions of the occipital cortex (homonymous or lateral hemianopsia). Hemianæsthesia and hemichorea, which have also been attributed to lesions of the posterior part of the thalamus, are probably the result of pressure on the sensory tracts in the internal capsule.

Lesions of the external capsule or claustrum (vide Figs. 103 and 104, 6 aek, 7 cl) cannot be diagnosed during life. They may exist without symptoms.

The symptoms of disease of the cornu Ammonis are unknown.

Lesions of the corpora quadrigemina have been associated with disturbances of sight, paralysis of the ocular muscles, and disturbances of equilibrium, but the number of authentic cases is small. There is reason for believing that the anterior and posterior corpora quadrigemina possess different functions. Destruction of the anterior pair may be assumed to produce amblyopia or amaurosis, with negative ophthalmoscopic appearances and absence of reaction in the pupils. Hemianopsia may be expected if the lesion is unilateral. Paralysis of branches of the motor oculi nerve (sometimes bilateral despite a unilateral lesion) has been observed in injury to the posterior pair. Symptoms of cerebral ataxia have also been attributed to this lesion.

Hitherto no characteristic symptoms of cerebellar diseases have been discovered. The cerebellar hemispheres may undergo extensive destruction without producing morbid symptoms during life. Nothnagel believes that diseases of the vermis are associated with staggering gait, cerebellar ataxia, and vertigo, but this view is contradicted by recent reports of cases.

Diseases of the cerebellar peduncles may run an entirely latent course. This is true of the anterior and posterior cerebellar peduncles, also known as the crura cerebelli ad corpora quadrigemina et medullam oblongatam. Lesions in the crura cerebelli ad pontem may give rise to symptoms if the connection with the cerebellum is not entirely interrupted and irritative effects are produced. Such lesions result in certain forced movements in which the body occasionally turns completely around its vertical axis, sometimes towards the side of the lesion, sometimes towards the opposite side. In Nouet's case, the eyes were immovable, the right eye being directed downwards and externally, the left eye upwards and internally. Simple lateral position of the body, and rotation of the head and eyes in the same direction (conjugate deviation) also occur in lesions of many other parts of the brain.

g. Focal Symptoms in Diseases of the Base of the Brain.

Morbid processes at the base of the brain are characterized particularly by implication of the cerebral nerves (compression paralysis).

These paralyses generally affect all the branches of the nerves, and

FIG. 108.
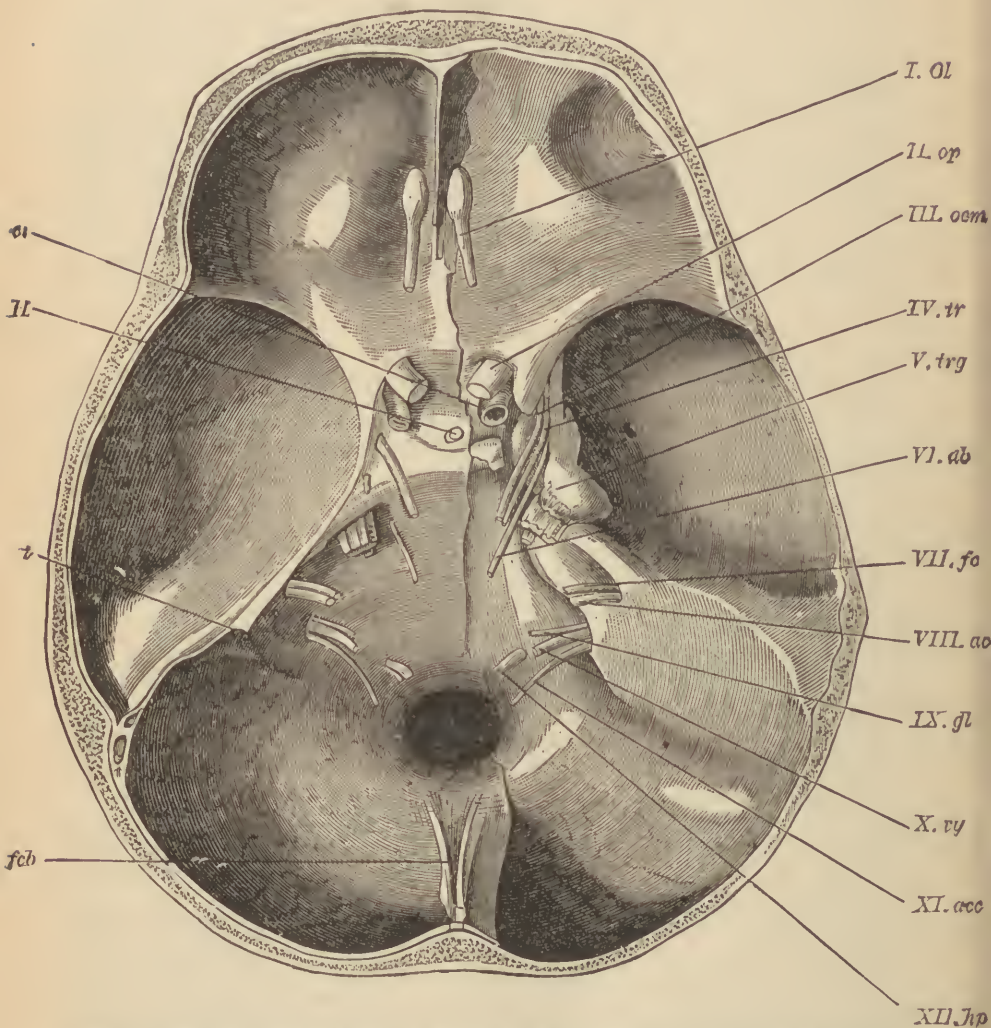
Base of the skull with emerging cerebral nerves. After Henle. I. *Ol*, olfactory nerve ; II. *op*, optic nerve ; III. *ocm*, oculo-motor nerve ; IV. *tr*, trochlear nerve ; V. *trg*, trigeminus nerve ; VI. *ab*, abducens nerve ; VII. *fc*, facial nerve ; VIII. *ac*, acoustic nerve ; IX. *gl*, glosso-pharyngeus nerve ; X. *vg*, vagus nerve ; XI. *acc*, spinal accessory nerve ; XII. *hp*, hypoglossus nerve ; *ci*, internal carotid artery ; *H*, hypophysis ; *t*, tip of the tentorium ; *fcb*, falx cerebelli.

have the electrical characteristics of peripheral paralysis. They are not infrequently bilateral. If the morbid process is located in the anterior fossa, the changes may be confined to the olfactory nerves, and manifested by loss of smell or perverse olfactory sensations (vide Fig. 108).

If the middle fossa is affected, the optic, oculo-motor, trochlearis, tri-geminus, and abducens nerves are involved (Fig. 108, V., VI.); in dis-eases of the posterior fossa, the facial, acoustic, glosso-pharyngeus, vagus, spinal accessory, and hypoglossus are implicated (Fig. 108, VII., XII.). Under the latter conditions, the symptomatology may be very similar to that of progressive bulbar paralysis. The morbid process spreads occasionally from one fossa to another, so that very remote nerves may become affected. In diseases of the posterior fossa, the pons and medulla are often affected, and this results in paralysis of the limbs. Implication of the cerebellum may give rise to the symptoms of cerebellar ataxia.

h. The Aphasic Symptom-Complex.

(Aphasia, Agraphia, Alexia, Amimia, Apraxia, Asymbolia.)

1. The term aphasia is applied only to disturbances of speech, but these are often associated with disturbances in the ability to write (agraphia), to read (alexia), or to express ideas by means of gestures (amimia), so that the patient may have lost the power of communicating ideas to his fellow-man or of understanding those of others. The pa-tients have lost the facultas signatrix (Kant), they suffer from asymbolia (Finkelnburg) or asemia (Steinthal).

There are various forms of aphasia, agraphia, alexia, and amimia, and various combinations of the individual forms.

2. In aphasia, the most important and striking symptom is the use of wrong words by the patient. Wernicke first showed that there are two principal varieties of aphasia, according as there are disturbances in the mechanism of speech or in the perceptive part of the faculty of speech. The former is known as motor or ataxic aphasia, the latter as sensory aphasia.

The child learns to speak by hearing words and attempting to imitate them, without associating the words, at first, with any definite idea. The acoustic nerve of the child receives the word, spoken by others, con-ducts it to its centre in the cerebral cortex (acoustic centre), whence other conducting paths convey it to the centre of the speech muscles, and thence, after a while, it gives rise to volitional or co-ordinated movements of the speech muscles innervated by the speech centre. This process is shown schematically in Fig. 109.

The child gradually emerges from the stage of an automatic speak-ing-machine to that of ideational speech. It learns to associate with the word a certain idea, and in this way becomes able to understand others and to make its own thoughts understood by others. In addition to the cortical centres for the acoustic and the speech muscles, there also develops an ideational centre which converts the speech image conveyed from the acoustic nerve to the cortical acoustic centre into a conception, and furthermore, by its connection with the cortical speech centre, con-verts ideas into words. This process is shown schematically in Fig. 110.

a. In cases in which the cortical speech centre is disturbed, the pa-tients understand everything that is said to them. They protrude the tongue when directed to do so, point to this or that object as required—in other words, present no disturbances in the perceptive or ideational part of speech. A different state of affairs is manifested when they are directed to repeat words or to clothe their own ideas in language. Dis-

turbances then make their appearance, because the muscles of speech, as the result of changes in their cortical centre, have lost the power of obeying the will and of acting together in a co-ordinated manner. In advanced cases, the patient is unable to repeat or to utter voluntarily a spoken word. Sometimes his entire vocabulary is lost, with the exception of a few words or syllables or incoherent sentences, which are constantly repeated on attempting to speak. Cases of this kind are known as motor or ataxic aphasia.

b. In disturbances of the cortical acoustic centre, the patient hears the spoken word as an element of sound, and is by no means deaf, but the word-image is lost, and he is, therefore, unable to understand the spoken language of others. He does not perform any required action,
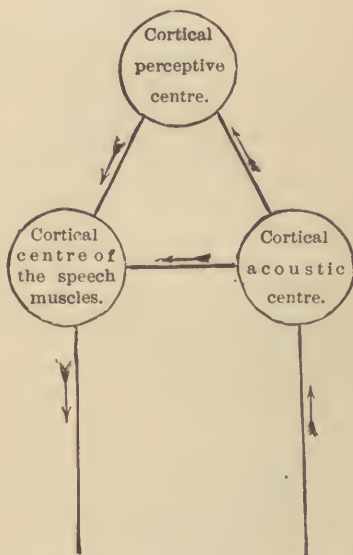
Fig. 110.

Fig. 109.
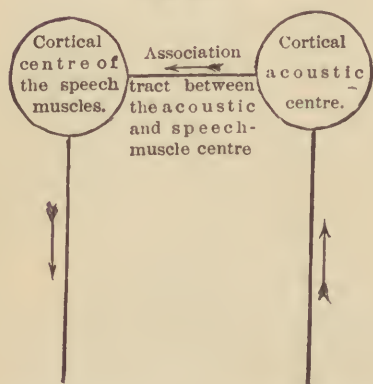
Speech-muscle tract.   Acoustic tract.   Speech-muscle tract.   Acoustic tract.

Schematic representation of the processes carried on in learning to speak.

Schematic representation of the processes carried on in the fully developed power to speak.

because he is unable to comprehend the spoken request. In examining the patient, care must be taken to avoid the use of gestures, since he may then perform the required action without comprehending the spoken request. Since the connection between the acoustic tract and the cortical speech centre is also interrupted, the patient is unable to repeat a spoken word. On the other hand, he is able to express his own thoughts in language. Kussmaul applied to this form of disease the term word-deafness. It is a variety of sensory aphasia.

c. When the functions of the cortical speech-muscle centre and of the acoustic centre are both impaired, total aphasia is produced. The patients are then unable to understand spoken words, to repeat them, or to speak voluntarily without hindrance.

As a matter of course, aphasic disturbances will be produced, not alone by diseases of the cortical speech-muscle and acoustic centres, but also by interruption of the conducting paths between these centres. These forms are known as conduction-aphasia.

d. In interruption to the conducting paths between the cortical speech-muscle centre and the ideational centre (vide Fig. 110, 1 S—3 B) the patient understands spoken words, is able to follow our directions and can repeat spoken words, but is unable to find words wherewith to express his own thoughts. Cases of this kind are known as amnesic aphasia.

e. In interruption to the conducting paths between the cortical acoustic centre and the centre of ideation (vide Fig. 110, 2 Ac—3 B), the patient can repeat spoken words and can also speak voluntarily, but he fails to understand the words which he hears.

Fig. 111.

Convolutions of the island of Reil (J. R.), after lifting up the operculum and adjacent convolutions.

f. In destruction of the conducting paths between the cortical acoustic and speech-muscle centres, the patient understands spoken words and can speak voluntarily, but the power of repeating words is interfered with. The intact condition of the centre of ideation may eliminate this defect, and paraphasia is then generally produced, *i. e.*, the patients often use the wrong words in voluntary speech.

g. Interruptions in the speech-muscle tract which emanate from the cortical speech-muscle centre will produce, so far as purely aphasic disturbances are concerned, the same symptoms as an affection of the the muscle centre itself, *i. e.*, the patients understand spoken words, but are unable to repeat them, and cannot speak voluntarily without hindrance. Since these paths must be looked for in the white matter adjacent to the cortical speech-muscle centres, it appears to be impossible during life to distinguish purely aphasic disturbances produced by a cortical lesion from those produced by a lesion in the adjacent white matter.

h. Disturbances in the tract of the acoustic nerve will produce apha-

sie symptoms similar to those resulting from destruction of the cortical acoustic centre itself, viz., inability to understand language, inability to repeat spoken words, but intactness of voluntary speech. The differential diagnosis between lesions in the cortical acoustic centre and the adjacent white matter is impossible.

3. Our present knowledge justifies the conclusion that the inferior (third) frontal convolution is affected in motor or ataxic aphasia, the superior (first) temporal convolution in word-deafness and, in all probability, also in amnesic aphasia; while the white matter adjacent to the island of Reil may be regarded as paths of association between the cortical speech centres of the inferior frontal and superior temporal convolutions. Aphasic symptoms occur only in lesions of the left side of the

Fig. 112.
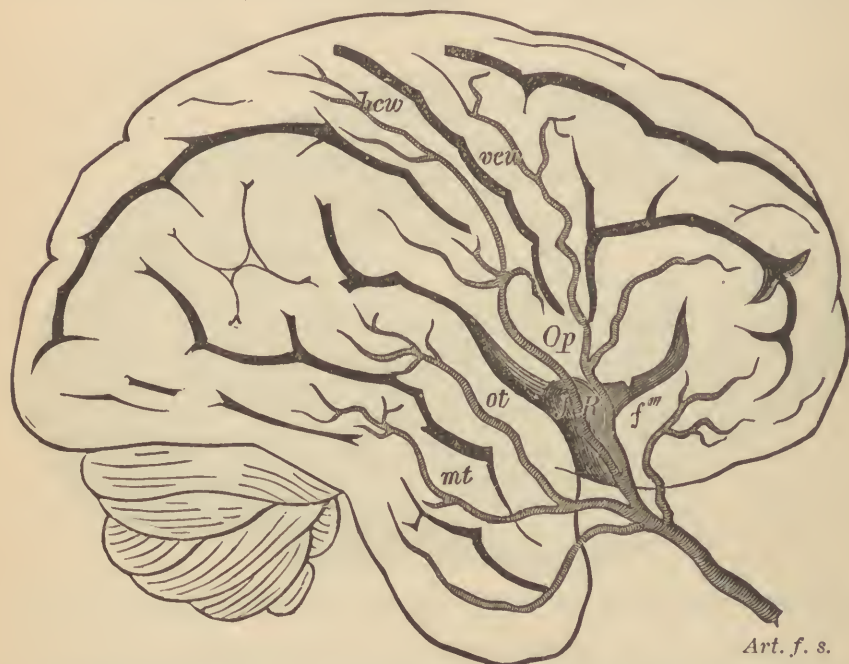
Distribution of the middle cerebral artery to the cortex of the cerebrum. Partly schematic. *Art. f. s.*, middle cerebral artery ; *f'''*, inferior frontal convolution ; *Op*, operculum ; *vcw*, anterior central convolution ; *hcw*, posterior central convolution ; *ot*, superior temporal convolution ; *mt*, middle temporal convolution. JR, Island of Reil.

brain, and are absent in right-sided lesions, unless the individual is left-handed.

The motor speech centre or the part involved in motor or ataxic aphasia does not include the entire third frontal convolution, but only the part immediately adjacent to the operculum (foot of the inferior frontal convolution) (vide Fig. 98). This part is nourished by the middle cerebral artery.

This artery also supplies the cortical acoustic centre in the superior (first) temporal convolution. The island of Reil, which is distinctly visible on lifting up the operculum (vide Fig. 111), is also supplied with blood by the middle cerebral artery (vide Fig. 112).

4. We have remarked on a previous occasion that the distribution of cerebral diseases, unlike those of the spinal cord, depends on the distribution of the blood-vessels. Hence changes in the cortical speech tracts are associated generally with diseases of the middle cerebral artery. Embolism or thrombosis of this vessel is the most frequent cause of aphasia. The peripheral branches of the artery are rarely affected, more frequently the main trunk of the vessel. Since this supplies not alone the third frontal convolution, first temporal convolution, and the island of Reil, but also the central convolutions (Fig. 112), aphasia is often associated with right hemiplegia, and the majority of cases are mixed forms.

Aphasia will also develop if the cortical speech centres are injured independently of the blood-vessels, for example, by direct injury, tumors, and inflammatory products of the meninges or bones. Congenital aphasia has also been observed (four cases in boys.)

As a rule, aphasia is a permanent condition which is capable of improvement or recovery only when other portions of the cortex become able, as the result of practice, to take part in the ability to generate speech.

Aphasic symptoms sometimes disappear gradually if they are not the result of destruction of the cortical speech centres, but of functional interference with them as the result of congestion, œdema, etc.

In addition, very brief aphasia has been observed in hysteria, chorea, catalepsy, after epileptic seizures, in helminthiasis and coprostasis. In such cases we must assume temporary circulatory disturbances without permanent anatomical lesions.

The prognosis and treatment depend on the primary disease. The aphasia itself may be treated by speech exercises which, if carried out persistently, sometimes produce rapid and marked success. Aphasic patients have been known to have the power of speech suddenly restored as the result of violent emotions.

5. Diseases of the cerebral cortex and adjacent white matter may also interfere with the ability to read, write, or to express ideas by means of gestures. It is not astonishing that aphasia should often be associated with agraphia and alexia. It is evident that writing from dictation is only possible when the cortical acoustic centre is intact, since otherwise the word-image could not be formed. Hence destruction of the cortical acoustic centre will also cause inability to write according to dictation. In disturbances of the visual centre the patients become unable to copy printed words. The muscles employed in writing, like the speech muscles, can be stimulated to co-ordinated activity from a certain centre; if this is destroyed, the patient suffers from motor or ataxic agraphia. If the paths of association between the ideational centre and the cortical centre of the muscles of writing are destroyed, the patient is unable to write voluntarily, he suffers from amnesic agraphia.

With regard to alexia it is known that the occipital lobes contain the cortical terminations of the optic nerve. If they are destroyed, the patients suffer from a form of alexia which corresponds to word-deafness. They are able to see the written characters, but the sign images are lost, the patients have become psychically blind. It is evident that this cortical centre of the optic nerve is connected by fibres of association with the cortical speech-muscle centre, else it would be impossible to read written characters aloud. Hence, in destruction of the cortical speech-muscle centre, motor alexia is present in addition to motor aphasia, and

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the patients are unable to read aloud, although they understand what has been read.

Psychical blindness does not always affect written characters alone. Cases are known in which the patients looked upon objects in every-day use as things unknown. When the patients were disrobed and the articles of clothing were then returned, they did not know to what uses to put them. This condition has been termed apraxia.

Finally, the patient may lose the ability to express himself by means of gestures (amimia). Sometimes they do not understand the gestures of others, sometimes they are unable to express their own ideas by means of gestures, either because the cortical centre for the co-ordinated activity of the mimic muscles is destroyed, or because the connection between the cortical centre of conceptions or ideas and the cortical centre of mimic movements is interrupted.

In all these conditions, the intelligence may be unimpaired, so that some patients carry on business in an intelligent manner, despite the existence of marked aphasia.

A. DISEASES OF THE SUBSTANCE OF THE BRAIN.

1. *Anæmia of the Brain.*

I. ANATOMICAL CHANGES.—Anæmia of the brain can hardly be separated from anæmia of the meninges. This is particularly true of anæmia of the brain and pia mater; the latter, as is well known, conveying blood-vessels to the brain. We distinguish between general and partial or circumscribed cerebral anæmia.

The condition is characterized anatomically by unusual pallor of the brain substance. The white substance has lost its pale rose-colored appearance and has assumed a dull white, occasionally bluish-white color (especially in children). The puncta vasculosa are absent or present in small numbers. The cortex has a pale-gray color, and the boundary between it and the subjacent white matter is often indistinct. The consistence of the brain varies; it is sometimes abnormally firm and dry, sometimes unusually succulent. This depends chiefly on the etiology, the former condition being observed when the anæmia is the result of extensive losses of blood, the latter when it follows œdema of the brain. The sulci on the surface of the brain are sometimes unusually broad.

Golgi claims to have found distention and unusual filling of the perivascular lymph spaces—perhaps a vicarious dilatation, to take the place of the narrow vessels.

The cerebro-spinal fluid is not infrequently increased in the subarachnoid tissue and the cerebral ventricles. The meninges are generally pale, and the sinuses sometimes contain very little blood. In some cases the meningeal vessels are markedly distended, compared with the pale cerebral parenchyma.

II. ETIOLOGY.—Cerebral anæmia is the result of changes in the blood itself or in the blood-vessels. The former include acute and chronic diminution of the amount of blood. Acute diminution occurs not infrequently after profuse losses of blood in any part of the body. In all such cases, other organs also take part in the anæmic condition.

Acute general anæmia of the brain sometimes develops on account of the vigorous efflux of blood to other parts of the body. For example, this has been observed after the puncture of ascites, ovarian tumors, or pleurisy. This may also occur after delivery when the uterus, freed of its contents, becomes engorged with blood. According to H. Fischer, the symptoms of shock are also the result of cerebral anæmia; this writer claims that in shock there is paralysis of the abdominal sympathetic, and therefore distention of the abdominal vessels supplied by it. Symptoms of cerebral anæmia also occur at times in individuals, especially old people, when an evacuation from the bowels takes place after long-protracted constipation.

Somewhat similar conditions are produced by affections which diminish the number of red blood-globules in the blood, or change their character in such a manner that the final effect is equivalent to that of anæmia. As a rule, the symptoms of cerebral anæmia run a chronic course in such cases. They include chlorosis, progressive pernicious anæmia, leukæmia, cachexia, suppuration, vital losses of all kinds.

Cerebral anæmia includes the symptom-complex known as hydrocephaloid, which is observed not infrequently in children, particularly nurslings, after exhausting diarrhœa. Signs of cerebral anæmia are observed not infrequently in cancer of the œsophagus or stomach; also after typhoid fever, relapsing fever, pneumonia, and other febrile infectious diseases, if inanition has been produced by fever and imperfect nutrition.

Cerebral anæmia from changes in the blood-vessels occurs in embolism and thrombosis of the cerebral arteries, but these conditions are usually followed very rapidly by the signs of necrotic (anæmic) softening of the brain. They will be discussed in a subsequent section.

Stenosis or even occlusion of the vessels is sometimes produced by compression from the outside, for example, by meningeal hemorrhages, exudations, tumors, or œdema of the brain. The compression is sometimes produced from within, for example, in dropsy of the ventricles. Tumors, aneurisms, exudations, arterio-sclerotic and endarteritic changes outside the skull may also give rise to compression of the vessels.

Cerebral anæmia is sometimes the result of weakness of the heart, for example, in fatty heart, pericarditis, and aortic stenosis.

Finally, stenosis of the cerebral vessels may be produced by vasomotor spasm, as the result of mental emotion or pain. Certain toxic agents may produce spasm of the cerebral arteries, but, as a rule, they also give rise to other symptoms which complicate the symptomatology.

III. SYMPTOMS.—It is not astonishing that the substance of the brain should react to anæmic conditions with functional disturbances. But the purely anæmic symptoms are often complicated by another set of phenomena. It has been shown by experiments that anæmia of the brain in animals lowers the pressure within the skull. It is true that the increased flow of cerebro-spinal fluid to the brain and the greater distention of the perivascular lymph spaces counteract the diminution of the cerebral pressure, but this can take place only to a certain extent, beyond which diminution of cerebral pressure with its sequelæ must ensue.

Sudden cerebral anæmia as the result of mental emotions, i. e., from spasm of the cerebral arteries, is attended by the following symptoms: the patients experience a sensation of oppression and anxiety in the chest, especially in the præcordial region, and occasionally there is tem-

porary palpitation of the heart. The patients yawn involuntarily, feel chilly, have a goose-skin, and look pale. There is impairment of hearing and ringing in the ears; sight is impaired until complete amaurosis develops; vertigo and nausea set in; consciousness becomes more and more clouded, and finally the patients fall with a low cry or twitchings in the face and limbs. This condition is known as fainting or syncope. Complete recovery occurs at the end of a few seconds or minutes. In some cases, however, syncope is followed by immediate death.

If cerebral anæmia is the result of acute losses of blood, the symptoms are very like those produced in animals by ligature of both carotid and vertebral arteries. The symptoms mentioned above are then associated with epileptiform convulsions, which remain absent only in individuals who are very much reduced.

According to Marshall Hall, the symptoms of hydrocephaloid are divided into two stages: In the first stage, that of irritation, the face is reddened, the eyes glistening and staring, the patient restless and fretful, sleep poor and disturbed; older children are delirious. In the second stage, that of torpor, the face is pale and pinched, the skin cool, fontanelles depressed, the lids half closed, the pupils wide, and their reaction slow or abolished, the back of the neck is rigid; the patients are in a condition of apathy, and suffer from convulsions. If no improvement occurs, the coma deepens, the pulse becomes imperceptible, the respirations irregular and feeble, and finally death ensues.

The symptoms of cerebral anæmia consist of irritative and paralytic phenomena which are often associated with each other.

The psychical functions are very often disturbed, probably as the result of anæmia of the cerebral cortex. Delirium often develops, sometimes very suddenly, and not infrequently assumes a violent and maniacal character. It may subside very rapidly, or it may last for days, or even weeks. Delusions may arise and in rare cases persist permanently. Some patients often suffer from obstinate insomnia (agrypnia), others from apathy, somnolence, or coma.

Disturbances of special sense are often observed; ringing in the ears, difficulty of hearing, flashes of light, impairment of vision, even amaurosis.

The patients often complain of headache, which has been attributed to anæmia of the dura mater; it is either unilateral or bilateral. Not infrequently there is violent vertigo. Complaint is sometimes made of paræsthesiæ.

Spasms and paralyses are frequent symptoms of cerebral anæmia.

The respirations are not infrequently irregular, unusually profound or superficial; there is often subjective and even objective dyspnœa.

The pulse may be irregular, accelerated, or retarded. Nausea and vomiting are frequent symptoms. The integument is generally pale and feels cold, and is covered occasionally with cold perspiration. Chilly sensations and chattering of the teeth are often observed.

It is noteworthy that the symptoms may only be present or, at least, are increased when the patient assumes the upright position.

IV. DIAGNOSIS.—Since some of the symptoms are the same as those of cerebral hyperæmia, the diagnosis can only be made with certainty when the etiological factors are taken into consideration.

V. PROGNOSIS.—The prognosis depends upon the etiology and the intensity of the cerebral anæmia. It sometimes acts as the immediate cause of death. Kussmaul attributes prognostic importance to the con-

dition of the pupils. Immediately after the occurrence of acute cerebral anæmia the pupils contract, and this is soon followed by dilatation and loss of reaction. The return of the pupils to the normal condition is a favorable indication.

VI. TREATMENT.—Brilliant results may be attained by prophylaxis. This includes nutritious diet and tonic treatment in febrile and wasting conditions, avoidance or rapid checking of hemorrhages, rapid relief of infantile diarrhœa, etc. In addition, the attendants should be directed to prevent feeble patients from assuming the upright position. The use of the night vessel instead of a bed-pan may be especially dangerous to such individuals.

If the signs of acute cerebral anæmia have developed, the patient's head should be kept as low as possible. If syncope occurs, the forehead should be rubbed with eau de Cologne, irritating substances (ammonia, eau de Cologne) inhaled, the nasal mucous membrane tickled with a feather, the face and chest douched with cold water, the soles of the feet rubbed, the faradic brush applied to the skin, or, if necessary, artificial respiration performed, for example, by faradization of the phrenic nerve (vide page 31).

The medicinal agents include stimulants and tonics: wine, soups, eggs, ethereal tincture of valerian (ten drops given a few times at intervals of fifteen minutes), sulphuric ether (five drops on sugar, or one-half to one syringeful subcutaneously), camphor (gr. xv.; ol. amygdalar., 3 iij., one syringeful subcutaneously), etc.

Morphine (gr. ¼), paraldehyde (gr. 75), or chloral hydrate (gr. xxx.), often produce remarkably rapid results in cases of great mental excitement and insomnia.

The danger arising from profuse hemorrhage must sometimes be met by the transfusion of physiological sodium chloride solution.

In chronic cases the galvanic current has been recommended, either a longitudinal current through the skull, the anode high up on the back of the neck (vaso-motor centre), or galvanization of the cervical sympathetic.

2. *Hyperæmia of the Brain.*

I. ETIOLOGY.—Hyperæmia of the substance of the brain is always associated with a similar condition of the meninges. Cerebral congestion may be acute or chronic, general (in the majority of cases) or partial (circumscribed). It may be the result of an abnormal supply of arterial blood (active hyperæmia, fluxion, congestion), or of impeded outflow of venous blood (passive or stasis hyperæmia).

Active cerebral hyperæmia may be produced by unusually vigorous contractions of the heart, such as occur in excessive bodily activity (running, lifting, etc.). It may also be the result of emotional excitement which stimulates the heart to increased action. As a matter of course, its occurrence is favored by left ventricular hypertrophy, particularly the so-called idiopathic hypertrophy. The symptoms of cerebral congestion may also appear in cardiac hypertrophy, following cirrhosis of the kidneys, more rarely in the hypertrophy which is secondary to valvular lesions. This is more apt to occur in aortic insufficiency, because the cardiac hypertrophy then corresponds, as a rule, to the severity of the valvular lesion, and both lesions compensate one another. In all these cases the cerebral hyperæmia will be general.

This is also true of those conditions in which congestion of the brain is owing to the fact that large vascular areas are excluded more or less completely from the arterial circulation. This is observed in stenosis of the isthmus aortæ, in enlargements of the liver or extensive abdominal tumors which compress the abdominal aorta, and thus increase the arterial flow to the brain. Obstinate constipation and distention of the intestines with fæces and gases may act in the same manner. After ligature of one carotid the half of the brain on the other side will be congested.

Cerebral congestion sometimes takes the place of suppressed hemorrhages (suppression of the menses or hemorrhoidal fluxes).

Dilatation of the cerebral vessels may be the result of vaso-motor paralysis (starting from the cervical sympathetic), which may occur spontaneously, or from mental or physical exertion, or the action of certain poisons (alcohol, amyl nitrite).

General cerebral hyperæmia may also occur as a collateral arterial fluxion, for example, in facial erysipelas, diphtheria, parotitis, abscesses on the outer surface of the skull, etc.

Circumscribed arterial congestion of the brain often follows other diseases of the brain or meninges (meningitis, meningeal or cerebral hemorrhage, tumors, abscesses, etc.).

The causes of venous congestion of the brain may be located in the skull, the large veins, or the heart.

Thus, it occurs in thrombosis of the cerebral sinuses; in obstruction to the escape of blood from the brain owing to compression of the internal jugular or innominate veins by inflammation in the neck or mediastinum, tumors in these localities, aneurism of the aorta, etc.

The outflow of venous blood is often interfered with by valvular lesions of the heart, particularly mitral stenosis and insufficiency and tricuspid insufficiency; also by diseases of the heart muscle (fatty degeneration, development of cicatrices), and by pericarditis (from pressure on the vena cava or implication of the heart muscle).

Venous congestion of the brain is also observed not infrequently in diseases of the respiratory organs (emphysema, interstitial pneumonia, pleurisy, chronic bronchitis, etc.). It is rare in phthisis, because this disease runs a very slow course, and is attended by a considerable diminution in the amount of blood.

Passive cerebral congestion may be produced voluntarily by continued straining. It is observed accordingly in diseases attended with cough, stenosis of the air passages, lifting heavy loads, straining at stool, suffocation, etc.

II. ANATOMICAL CHANGES.—The anatomical changes are not always easily recognized. They may be mistaken for post-mortem hypostasis, but this condition is confined to the most dependent portions of the brain. On the other hand, the symptoms of cerebral congestion may have been present during life, but nothing is found at the autopsy. In experiments on animals, Ackermann found that anæmia of the brain is present in suffocation, but Jolly showed that this occurs only at the moment of death, and that congestion of the brain is really present during life.

Cerebral congestion is shown by the increased amount of blood and the changed color of the brain. Upon removing the calvarium, numerous drops of blood exude from torn vessels on the outer surface of the dura mater and the inner surface of the skull. The Pacchionian

bodies are unusually well supplied with blood, and, in chronic cases, are often very large and numerous. The sinuses of the dura mater are often distended with blood, and contain loose, dark clots, rarely amber-colored or grayish-yellow clots. When the dura mater is cut horizontally, the brain often protrudes through the incision as if it had been cramped for room. Upon throwing back the halves of the dura mater, the large veins of the pia mater (in venous congestion) are found enlarged and distended, often sinuous; in some cases they are said to have presented varicose dilatations. The surface of the brain often appears flattened, the convolutions are flat and broad, the sulci narrow; the surface is often very dry. Opacities and thickenings are often visible on the arachnoid.

On section of the brain, the changed color of the parenchyma is particularly noticeable. The cortex is dark-red or brownish-red in appearance, while the white substance has a rosy red or hortensia color. The latter color is often especially marked in patches. Numerous dots of blood are present in the brain; in some cases small extravasations are found. The pigment in the latter may undergo changes, so that yellowish or rusty-brown patches result. The choroid plexuses are often distended with blood, and this is recognized mainly by the sinuosity of their vessels. The unusual congestion is also noticeable in the ventricles. The cut section of the brain is often unusually moist (œdema).

On microscopical examination an accumulation of red blood-globules is found not infrequently in the adventitious lymph sheaths of the blood-vessels.

In some places, the walls of the blood-vessels contain rents through which the red blood-globules have passed into the lymph sheath, and have distended the latter into an ampulla-shaped dissecting aneurism. If the congestion has lasted a long time, the red globules undergo gradual degeneration and metamorphosis of their coloring matter, so that the lymph sheaths are found filled with yellow pigment granules. These are especially abundant at the bifurcation of the vessels, because the adventitious lymph sheaths in that locality are very loose and present a larger space.

Gogli states that the perivascular lymph sheaths are narrow and empty. This indicates that an increase in the amount of blood within the skull is only possible if the lymphatic spaces in the brain have emptied their contents into the spinal cord and peripheral lymphatics. This also includes the displacement of the cerebro-spinal fluid into the spinal canal. Some authors state that chronic cerebral congestion results in overfilling of the ganglion cells with yellow pigment and increase of the neuroglia in the immediate vicinity of the blood-vessels.

Atrophy of the brain is regarded as a sequel of chronic cerebral hyperæmia, but in some cases the former condition is primary, the latter secondary. Durand-Fardel also attributes the so-called état criblé to cerebral congestion. It consists of dilatation of the perivascular lymph spaces, so that the blood-vessels are surrounded by gaping openings, whose size may exceed the dimensions of the head of a pin.

III. SYMPTOMS.—The symptoms of cerebral hyperæmia depend not alone on the changed nutritive conditions, but also upon changes of pressure. Jolly showed experimentally that the cerebral pressure is increased in venous hyperæmia as the result of ligature of the veins.

The symptoms of cerebral congestion are very similar to those of anæmia, and the diagnosis can sometimes be made only by taking the etiology into consideration. Irritative and paralytic conditions of the brain are produced in both cases.

The conditions differ according as the congestion is arterial or venous. In the former event, there is an increased supply of nutritive

material; in the latter, there is also poisoning with carbonic acid. Symptoms of irritation predominate in arterial congestion, symptoms of depression in venous congestion.

Disturbances of the psychical functions are very frequent, manifested by an irritable, whimsical mood and unusual excitability, or by an apathetic condition. The patients are unable to think clearly, they appear forgetful, and are often tortured by the fear of approaching insanity.

Delirium occurs not infrequently, and illusions, hallucinations, and maniacal attacks are also observed. If such symptoms last for a long time or recur at short intervals, the condition will lead to well-marked psychopathy.

Sleep is often disturbed and interrupted by frightful dreams; other patients are somnolent and dull. Many suffer from dizziness; at times from attacks of heat and rush of blood to the head, during which all the other symptoms increase in severity. Such attacks may terminate in complete unconsciousness.

Frequent complaint is made of headache. The pain is generally dull and diffuse, more rarely it is unilateral or confined to circumscribed parts of the skull.

The nerves of special sense, particularly of the eye and ear, are often unduly excitable, so that the patients are annoyed to an unusual degree by bright light, noises, etc. In other cases, they complain of flashes of light before the eyes, dimness of sight, ringing in the ears, etc.

Some patients complain of paræsthesiæ: feeling of deadness in the skin, formication, etc. These sensations may be unilateral, confined to one limb or to parts of a limb.

Muscular twitchings are not infrequent; they may be confined to a few muscles of the face or limb, or they may spread to an entire limb, to one side of the body, or become general; they are often associated with disturbances of consciousness and assume an epileptiform character. The differential diagnosis between epilepsy and cerebral hyperæmia not infrequently is very difficult.

Some patients complain of a dead feeling in the muscles, and pareses and paralyses may be present. As a rule, these are ephemeral and disappear as soon as the circulatory disturbance is relieved.

It has always been held that puffy, short-necked individuals, who live well, take little exercise, i. e., plethoric individuals, have a special tendency to cerebral congestion; but lean, slender individuals may also suffer from this affection.

During the attacks, the face is often very red, in passive congestion it is intensely cyanotic. The heart's action is not infrequently accelerated, stronger, occasionally irregular; the patients often complain of palpitation and an anxious feeling of oppression in the præcordial region. In active cerebral hyperæmia, the pulse feels full and hard, in the passive form it grows small, even imperceptible. The carotids pulsate vigorously, and pulsations are sometimes visible in the smaller arteries. These changes may be associated with irregular respirations. During apoplectiform and comatose attacks, the breathing becomes stertorous and may even assume the Cheyne-Stokes type. Slight rise of temperature is not unusual.

Vomiting must be regarded as a symptom of cerebral irritation. Involuntary evacuations sometimes occur during comatose conditions.

The symptoms mentioned above may appear in various degrees of intensity, combination, and duration. They sometimes last only a few

seconds, sometimes hours, days, weeks, even months. Relapses occur in very many cases.

We distinguish four characteristic groups of symptoms.

a. Cephalalgic form. The headache, feeling of confusion in the head, and hyperæsthesia to light and sound predominate. In addition, palpitation of the heart, rush of blood to the head, etc.

b. Psychical form. The patients are excited and sleepless, suffer from illusions and hallucinations, become maniacal in unfavorable cases, and may become permanently insane.

c. Convulsive form. This is observed most frequently in children. It is characterized by twitchings and epileptiform attacks, often by tetanic stiffness of the muscles of the back of the neck. It may be mistaken for epilepsy, meningitis, or tetanus.

d. Apoplectic form. Attacks of unconsciousness occur in this form as in cerebral hemorrhage. With or without prodromata, the patients fall unconscious to the ground, remain comatose for a longer or shorter period; and on coming to, present pareses and paralyses which soon disappear. Experience teaches that these symptoms not infrequently precede cerebral hemorrhage.

IV. DIAGNOSIS.—The diagnosis is generally easy if we take into consideration, not alone the symptoms, but also the causes of the affection. As a rule, the latter will also determine whether the hyperæmia is active or passive.

V. PROGNOSIS.—The prognosis is often serious or unfavorable on account of the primary affection. If the causes can be removed, the prognosis is generally good, although death may occur during a convulsive or apoplectiform attack.

IV. TREATMENT.—Causal treatment varies according to the primary affection—digitalis in heart disease; restricted diet, exercise, etc., in plethora, etc.

In active cerebral congestion, the upper part of the body should be kept elevated, from eight to sixteen ounces of blood removed by venesection (in children four to six leeches to the mastoid processes), an ice-bag or cold applications applied to the head, and the bowels thoroughly opened. Hot foot-baths or mustard hand-baths may be employed to produce a rapid derivative effect. The food should be fluid, the room should be large and airy and a little darkened; absolute bodily and mental rest. Narcotics should not be given if the patient suffers from insomnia; potassium bromide (℥ i.–iiss. at night) alone may be recommended. The after-treatment may consist of a cold-water cure or a trip to the mountains or sea-shore. Chronic congestion has been treated with the galvanic current (longitudinal current through the skull, anode upon the forehead, cathode on the back of the neck near the vaso-motor centre).

3. Œdema of the Brain.

In cerebral œdema, the cut surface of the substance of the brain appears unusually moist. Its consistence is generally diminished and sometimes described as almost diffluent, but such cases are often the result of post-mortem maceration. In many instances, the brain substance is unusually pale; its volume may be increased and the surface appear flattened. Not infrequently there is increase of the cerebro-spinal fluid in the ventricles and subarachnoid space, and also œdematous swelling of the subarachnoid meshed tissue.

The microscope is said to show dilatation and overfilling of the perivascular lymph spaces.

Cerebral œdema is so much more apt to interfere with the functions of the brain the more rapid and diffuse the development of the œdema. It produces the symptoms of increased cerebral pressure, so that in individual cases it re-

mains for us to ascertain the causes of the condition (hemorrhage, hyperæmia, tumor, etc.). In many cases cerebral œdema is an agonal phenomenon.

The difficulty of arriving at a correct conclusion concerning œdema of the brain is evident from the differences of opinion concerning its relations to uræmia.

The causes of cerebral œdema are the same as those of œdema in other organs —circulatory disturbances and changes in the constitution of the blood, with secondary increased permeability of the walls of the vessels.

4. *Cerebral Hemorrhage.*

(*Encephalorrhagia.*)

I. ETIOLOGY.—Primary or spontaneous cerebral hemorrhage, which will alone be considered, generally occurs beyond the age of 40 years, though cases are also observed in childhood. Its relative frequency increases with each decade beyond the age of 40 years.

It is more frequent in men than in women. Hereditary influences are sometimes demonstrable.

Experience teaches that it is more frequent during the winter months.

The development of cerebral hemorrhage depends on three possibilities: excessive blood pressure in the arteries, disease and diminished resistance of the cerebral vessels, and, finally, cerebral changes in the immediate vicinity of the blood-vessels (softening and atrophic disappearance).

It is not probable that the mere increase of arterial pressure will give rise to a hemorrhage into the brain. It has been proven, experimentally, that rupture of the cerebral arteries requires a pressure which is so enormous that it can hardly ever be produced during life.

The opinion is constantly growing stronger that changes in the blood-vessels (with diminished resistance) are always the cause of cerebral hemorrhage, and the theory that rupture of the vessels may result from softening or atrophy of the surrounding brain substance is now universally abandoned. The latter event is perhaps possible, but at all events it is exceptional.

The changes in the vessels, which consist generally of the formation of miliary aneurisms in the smaller cerebral arteries, produce, *per se*, the tendency to rupture. But the latter will be apt to occur so much sooner if the arterial blood pressure is also increased. In some cases, the hemorrhage occurs during sleep; in others, immediately after some event which increases the arterial pressure (anger, mental excitement, bodily exertion, a heavy meal, etc.). Special mention should be made of the fact that hypertrophy of the left ventricle with secondary increase of the blood pressure in the aortic system is a frequent exciting cause of cerebral hemorrhage. This is less true of hypertrophy following valvular lesions—the hypertrophy then merely compensates the valvular lesions— but rather of the hypertrophy following cirrhosis of the kidneys and arterio-sclerosis. It was also mentioned in the paragraph on congenital stenosis of the isthmus aortæ that an unusually large proportion of the patients die of cerebral hemorrhage (vide Vol. I, page 170).

Stasis in the venous system sometimes acts as an exciting cause of hemorrhage of the brain, for example, as the result of violent straining at stool, lifting a heavy load, violent coughing, sneezing, laughing, in rare cases from straining during delivery, etc.

II. ANATOMICAL CHANGES.—If the hemorrhage has attained certain

dimensions, its location can sometimes be recognized after removal of the dura mater. The brain projects more markedly on the affected side, the gyri are flattened, the sulci flat, the brain is often anæmic from compression of the vessels. In some cases, the falx cerebri is pushed over strongly towards the unaffected side. At times the hemorrhage is not confined to the parenchyma, but passes into the ventricles or breaks through the cortex and pia mater, and may enter the subarachnoid space in both directions. It then spreads to a considerable extent over the surface of the brain, and may even surround the larger part of the circumference of the brain like a sort of bloody cap. The blood may pass likewise into the subarachnoid cavity of the spinal cord, and extend far down the spinal canal. It is generally coagulated, and may be removed readily from the subjacent parts.

The size of the hemorrhage is extremely variable; it sometimes occupies an entire hemisphere. In some cases, more than twelve ounces are extravasated. The hemorrhage may be round, elongated, or irregular in shape. As a rule, hemorrhages in the white matter are less extensive and more elongated, because the white matter offers greater resistance to the extravasated blood than the gray matter. If the hemorrhage affects both parts at the same time, it is generally more extensive in the gray matter.

In the majority of cases, it takes place into one hemisphere alone, more rarely it is bilateral. Hemorrhages in the vicinity of the median line of the pons are apt to involve both halves of that organ. A number of hemorrhagic foci, varying in age, are found not infrequently.

In recent cases, the hemorrhagic focus forms a soft mass which consists of extravasated blood and débris of brain tissue. At the periphery of the hemorrhage the brain forms a ragged, floating fringe, from which coils of vessels sometimes pass into the extravasation. The parts around the latter are often infiltrated with punctate hemorrhages, appear swollen, moist, yellow (citron-colored œdema). .

Next the blood coagulates, and then the red blood-globules gradually degenerate, and their blood pigment becomes changed and crystallizes. The color of the clot changes from chocolate to rusty brown, finally to ochre yellow, the extravasated blood is gradually absorbed and is replaced by serous fluid. In the mean time, reactive inflammation has occurred in the adjacent layer of brain tissue, the principal part being played by the neuroglia. Connective tissue is produced and encysts the hemorrhagic focus (apoplectic cyst).

The contents of an apoplectic cyst are not always clear and serous. In some cases they form a milky, emulsive fluid, on account of the abundant mixture of fat drops and granulo-fatty cells. Blood crystals, in the shape of a brick-red or brownish-red precipitate, are especially apt to be found on the inner surface of the cyst-wall. In rare cases the cyst exceeds the size of a walnut, and may even attain the dimensions of an apple.

The reactive inflammation around the hemorrhagic focus is sometimes so active as to give rise to extensive encephalitis. Under such circumstances the cysts may have purulent contents. In rare cases, non-encapsulated cysts are found.

The cavity of the cyst is sometimes traversed by fine threads, which consist in part of persistent blood-vessels, in part are newly formed. Their number may be so considerable as to form a multilocular, narrow-

meshed cavity whose chambers are filled with fluid.   It is supposed that
the cysts may disappear, from gradual absorption of the fluid and the
approximation of their walls, so that finally nothing is left but a pig-
mented, apoplectic cicatrix.   Apoplectic cysts and cicatrices may pro-
duce torsion upon adjacent parts of the brain.

In hemorrhages into the cortex, the pia mater becomes thickened

FIG. 113.

Miliary aneurisms on a small artery in the lenticular nucleus.   Enlarged 25 times.   After Marchand.

above the site of hemorrhage.   Beneath it is found a serous, apoplectic
cyst or a depressed apoplectic cicatrix.

Microscopic examination of recent hemorrhagic foci reveals red blood-globules,
débris of nerve tissue, amœboid cells and granulo-fatty cells.   There are often a
variable number of cells containing blood-globules (ingestion by amœboid cells).
Next the red globules undergo shrinking and granular disintegration, and the
number of granulo-fatty cells increases.   The free pigment stains the other struc-

FIG. 114.
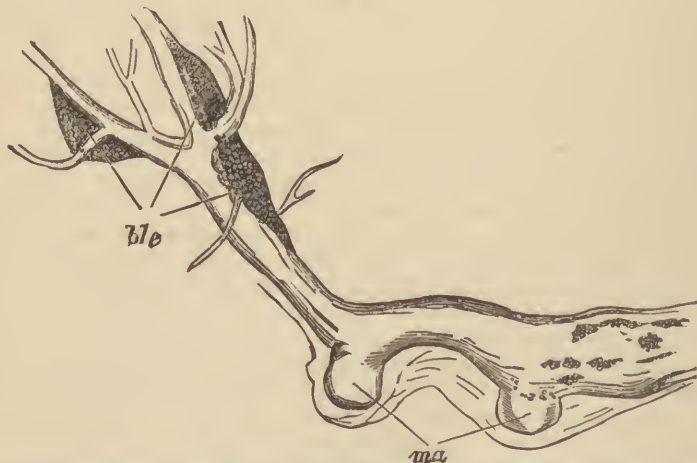
Artery from an apoplectic focus.   *ma*, miliary aneurisms ; *ble*, extravasations into the adventi-
tious lymph spaces.   After Cornil and Ranvier.   Enlarged 30 times.

tures yellow, in part it is deposited free or in the cells in the form of granules,
needles, or tablets; it is particularly abundant upon the inner surface of the cyst-
wall.   The remains of the vessels are very fatty, and hæmatoidin granules or
crystals are found not infrequently in their lymph sheaths or on their outer sur-
face.

The changes in the blood-vessels are best detected by removing the hemor-
rhagic focus, in addition to the adjacent brain substance, placing it in water,
carefully renewing the water during the next few days, and then cautiously
pouring a stream of water over the brain substance, until nothing is left but the
vessels.   The latter are then removed with forceps and placed upon a glass slide.

The vascular changes, so-called miliary aneurisms, affect only the smaller vessels, and are often visible to the naked eye. They may be one millimetre in size (vide Fig. 113), or recognizable only with the microscope. They are apt to develop at the point of the bifurcation of the arteries, and are often present in large numbers.

At the aneurism, the muscular coat of the vessel has disappeared, and the intima and adventitious lymph sheath approach one another until they come in contact. In some places, the aneurism is found ruptured, and the blood extravasated into the adventitious lymph sheath (vide Fig. 114, *ble*). In others, the latter contains a rent through which blood has passed into the surrounding parenchyma.

Bouchard and Charcot regard the aneurism as the result of periarteritis, which consists of increase of the nuclei in the adventitious lymph sheaths and thickening of the latter, and disappearance of the muscular coat. Zenker attributes the lesion to arterio-sclerosis. Finally, Roth maintains that the aneurism is the result of a primary affection of the muscular coat, which first undergoes hypertrophy, and causes diffuse cylindrical dilatation of the arteries; later, the muscular coat undergoes fatty and waxy degeneration. At the same time, the artery suffers aneurismal dilatation, which antagonizes the thickening of the adventitious lymph sheath or tunica intima. Although the aneurisms manifest a tendency to rupture, Roth believes that there is sometimes an attempt at recovery in the shape of thickening of the intima, which may advance to complete obliteration.

Miliary aneurisms are observed almost constantly in cerebral hemorrhage. Although the possibility of cerebral hemorrhage from fatty degeneration and simple atheromatous changes of the vessels cannot be denied, still this is undoubtedly exceptional.

Miliary aneurisms are particularly apt to be found in advanced life. They are most frequent in the optic thalamus and corpus striatum, next in the cerebral convolutions and the pia mater, then in the pons, medulla oblongata, and gray matter of the cerebellum. Cerebral hemorrhages are found most frequently in the optic thalamus and corpus striatum; then follow the other parts of the cerebrum, then the cerebellum, pons, and cerebral peduncle; next the medulla oblongata and corpora quadrigemina; the cornu ammonis, corpus callosum, and fornix almost always escape.

Hemorrhages into the ventricles are generally derived from foci which have broken through the thalamus, corpus striatum, or the pons. Rupture of a vessel in the wall of the ventricle occurs much more rarely.

Individuals who die soon after the occurrence of a cerebral hemorrhage, sometimes present hemorrhages in internal organs, for example, the bronchial mucous membrane, lungs, or kidneys, gastro-intestinal mucous membrane; if the organs are two in number, in the one opposite to the side of the cerebral hemorrhage. This has been attributed to vaso-motor disturbances.

In three cases, Lepine noticed that the paralyzed limbs grew cold more rapidly after death than the healthy limbs.

Liouville detected miliary aneurisms in the arteries of the retina.

After the hemorrhagic foci have existed for a long time, they are followed by secondary degeneration in certain systems of fibres in the central nervous apparatus (vide page 131).

Unilateral atrophy of the brain also occurs as a result of cerebral hemorrhage, especially into the cortex.

The peripheral nerves and muscles remain unchanged. But a few doubtful cases have been reported in which there was an increase of the interstitial tissue in the peripheral nerves. Meissner mentions degenerative changes in the tactile corpuscles, but this is contradicted by Langerhans. Debove mentions dilatation of the medullary spaces and Haversian canals of the bones on the hemiplegic side.

IV. SYMPTOMS.—In typical cases, cerebral hemorrhage presents five

stages, the prodromata, the apoplectic attack, the inflammatory reaction, the permanent symptoms, and the secondary changes.

The prodromata generally consist of the symptoms of cerebral congestion, viz., rush of blood to the head, vertigo, slight confusion, flashes of light before the eyes, ringing in the ears, a feeling of anxiety and oppression. The patient generally grows excitable and whimsical, and complains of restless sleep, interrupted by bad dreams. He often suffers from headache, which occurs either spontaneously or after bodily or mental exertion; the pain may be diffused or circumscribed. The patient feels incapable of mental work, the memory is impaired. In some cases there is occasional loss of words, more rarely articulation is impeded and the speech thick.

These symptoms may precede the apoplectic attack for months, weeks, days, or hours.

In six cases, Foerster observed small hemorrhages into the conjunctiva and retina of individuals who died several years afterwards of cerebral hemorrhage.

The symptoms become more serious when complicated with unilateral disturbances of motion or sensation: formication, prickling, weakness of the limbs, dragging of one leg in walking, etc. These symptoms are confined not infrequently to one limb, and may disappear at the end of a few hours or days. These are probably the result of small hemorrhages, which are capable of rapid reparation.

The apoplectic attack is either preceded for a longer or shorter period by the prodromata just mentioned, or it develops suddenly and unexpectedly. In the severest cases, the patients fall as if struck down; they are completely unconscious, do not react to irritation, pass urine and fæces involuntarily. Death may take place almost immediately after the onset of the apoplectic seizure (apoplexia fulminans s. attonita).

When the patient is profoundly comatose, it may be difficult to recognize the existence of a hemiplegia. The limbs are completely flaccid, and if raised fall like dead masses. Hence, the condition may be mistaken for drunkenness and opium poisoning. But drunken individuals will generally smell of alcohol, and in opium poisoning the pupils are contracted to pin-point dimensions.

As a result of the unilateral paralysis, changes are often noticed on one side of the face: flattening or abolition of the naso-labial fold, to and fro movement of the flaccid cheek during inspiration and expiration, flow of saliva from the open angle of the mouth, depression of one-half of the palatal arch, and deflection of the uvula (the latter is physiological in some individuals), sometimes towards the healthy, sometimes towards the paralyzed side.

Pinching or pricking the skin produces no reflex movements in profoundly comatose patients. Less comatose individuals contract the non-paralyzed side of the face, or groan and moan. In still slighter grades of coma, the limbs on the unaffected side are moved, those on the other side remain motionless. If the limbs are raised, those on the paralyzed side fall as if dead, those on the opposite drop slowly. Passive movements of the paralyzed side may sometimes be performed without resistance.

In profound coma, attention must also be paid to the presence or absence of certain cutaneous reflexes (reflexes of the testicle, of the muscles of the abdomen, loins, and buttocks, and of the nipple).

If the finger or other hard body is rubbed across the integument of the upper and inner part of the thigh, or if the saphenus major nerve is compressed about a hand's breath above the internal condyle of the femur in the space between the sartorins and vastus internus, the testicle on the side corresponding to the irritation will be elevated by the contraction of the cremaster muscle. Rapid stroking over the abdomen, buttocks, or loins will produce reflex muscular contraction upon the irritated side. Stroking of the nipple will give rise to erection of this part and to wrinkling of the pigmented areola.

In hemiplegics, on the other hand, these reflex movements remain absent on the paralyzed side, in recent cases as well as during the status apoplecticus. This symptom is so much more important because these reflexes are rarely absent in healthy individuals. In the further course of the disease, the reflexes gradually return, but are distinctly weaker, so long as the paralytic symptoms persist.

In some cases hemiplegia does not occur, but in its stead appear epileptiform, general or unilaterel convulsions, or unilateral contractures. This is especially true of cortical or pons hemorrhages, or those which have ruptured into the ventricles.

Some patients assume forced positions. The head and eyes are turned constantly to one side, sometimes the trunk is also turned in the same direction. If the patient is moved out of the forced position, he will gradually return to it. The rotation sometimes occurs towards the side of the paralysis, sometimes in the opposite direction.

During the apoplectic seizure, the face is often red and congested, the carotids throb violently, and there are often vigorous pulsations in the temporal arteries. The pulse is tense, slow, normal or accelerated, and not infrequently irregular. In some cases the patient is pale, the heart sounds feeble, and the pulse small. The respirations are often arhythmical, or Cheyne-Stokes breathing is observed. The respirations are often snoring and stertorous, and at each inspiration it is found that the paralyzed ala nasi closes the nostril and prevents the entrance of air. Tracheal râles will be heard if the saliva and mucus accumulate in the upper air passages. The pupils may be narrow or irregularly dilated, and destitute of reaction; the latter is a bad prognostic sign. In a case of apoplexy which developed under my observation, I noticed that the pupil upon the side opposite to the hemorrhage dilated, and assumed the shape of a cat's pupil.

The patient comes out of the apoplectic attack either quite suddenly, for example, after venesection, or days and weeks elapse during which consciousness gradually returns. If the disease terminates fatally, life is gradually extinguished after increasing disturbances of respiration, tracheal râles, cyanosis, and increasing general collapse.

Bourneville states that, in a quarter hour to a half-hour after the beginning of the attack, the bodily temperature sinks below the normal. If the case terminates favorably, the temperature returns to the normal after the diffuse cerebral symptoms have subsided, and the focal symptoms have become more distinct. A fatal termination is preceded for several days by a considerable rise of temperature. In apoplexia fulminans, the temperature continues to sink until the moment of death.

Ollivier makes the following statement with regard to the urinary excretion. Immediately (half-hour) after the hemorrhage, there is an increased secretion of urine, so that in one case two thousand cubic centimetres were passed in two hours. The urine is very pale and its specific gravity very low (1.004). It contains but little urea, some albumin, and later sugar. The albuminuria is at first

slight and then increases gradually, but all these changes disappear in twelve to
twenty-four hours. The urinary changes are so much more marked the more
profuse the hemorrhage, *i. e.*, the more violent the shock to the medulla oblon-
gata, to which Ollivier attributes these symptoms. Sudden increase in the
amount of urea excreted is an unfavorable prognostic event.

In some cases, the coma, at the period at the apoplectic seizure, is not
sufficiently profound to prevent movements of the limbs or manifesta-
tions of pain upon irritating the skin. The patients sometimes come
out of an apoplectic attack and remain in excellent condition for days;
then follows another seizure, perhaps more severe than the first, and the
patient succumbs.

The apoplectic seizure is sometimes the third stage of the disease, in-
asmuch as it is preceded by paralytic symptoms. Some time ago I had
under observation a man æt. 60 years, who suddenly fell, while hunting,
on account of paralysis of the right leg. I saw him two hours later; con-
sciousness was perfectly clear, but the right arm and leg could not be
moved. Somnolence developed thirty hours afterwards, and deepened
into coma of several days' duration.

In some cases, there is a so-called abortive form of apoplexy: vertigo,
vomiting, slight syncope, etc.

The chief cause of the apoplectic seizure is the sudden elevation of brain pres-
sure produced by the extravasation. The second factor is the purely mechanical
effect, the concussion or shock to the brain, whose intensity likewise depends on
the profuseness and rapidity of the hemorrhage. Another factor is the anæmia
of the brain resulting from the compression. Finally, it must not be forgotten
that the site of the hemorrhage also influences the development and mode of on-
set of the apoplectic seizure.

The symptoms of reaction are manifested by an increase of tempera-
ture (more that 2° C.), delirium, and renewed somnolence, often by slight
twitchings and contractures in the paralyzed limbs, occasionally by pain.
They generally begin from the second to the fourth day after the seizure,
and are the result of the inflammatory changes in the vicinity of the
extravasation.

Permanent focal symptoms develop so much more distinctly the
more the symptoms of the apoplectic attack and of reactive inflamma-
tion subside. In the majority of cases, they present a striking similarity
because the majority of hemorrhages affect the internal capsule in addi-
tion to the adjacent corpus striatum (and optic thalamus). We will con-
fine our remarks to the consideration of this principal type.

Hemorrhages present a predilection for the region in question for two reasons:
first, because miliary aneurisms are especially frequent in this locality; secondly,
on account of the relations of the blood pressure. The arteries supplying the
basal ganglia are given off directly from the anterior and middle cerebral
arteries and are therefore subject to the changes in blood pressure in the domain
of the internal carotid which would otherwise be enfeebled if there were nu-
merous arterial branches.

In hemorrhages into the internal capsule (and adjacent corpus stria-
tum) the chief symptom is motor paralysis of one side (cerebral hemi-
plegia).

Unlike peripheral and pons paralyses, the facial nerve is not affected
in all its branches. The frontal branch escapes, so that the forehead can
be wrinkled and the eye closed. The cheek and region of the chin on
the paralyzed side are motionless.

In explanation of the non-paralysis of the frontal branch of the facial nerve, some assume two distinct centres and separate central paths for the two parts of the facial nerve, others believe that both frontal branches are innervated by one cerebral hemisphere, so that in a unilateral lesion the other hemisphere acts vicariously.

The hypoglossus is more or less paralyzed. This is shown by impaired mobility of the tongue in mastication and speaking; the speech is slow and thick. The tongue, when protruded, deviates toward the paralyzed side (predominance of the non-paralyzed genioglossus muscle). In the further course of the disease, the hypoglossal paralysis disappears in part, but, as a rule, not entirely.

The paralysis of the arm is more marked than that of the leg, and it has been supposed that the prognosis is unfavorable if the reverse holds good.

The muscles of the thorax and abdomen also take part in the paralysis, so that the movements of respiration and straining are less vigorous on the paralyzed side. Paralysis of the trapezius is shown by the sinking of the shoulder.

Pitres and Friedlaender have recently detected impaired mobility of the limbs on the unaffected side (influence of the uncrossed anterior column-pyramid tract).

In a number of cases I have found, with the laryngoscope, impaired mobility of the vocal cord on the paralyzed side.

The following phenomena have been observed in the paralyzed limbs: a. Eulenberg noticed that the pulse upon the paralyzed side is smaller and presents a more marked elevation of recoil; b. According to Bricquebeck and Charcot, the blood taken from a vein on the paralyzed side looks redder than that taken from the opposite side; c. Lépine and Bricquebeck noticed that the paralyzed limbs are cooled more rapidly in cold water than the healthy ones; d. According to Charcot, the temperature of the paralyzed limbs increases a few hours after the seizure (sometimes as much as 9°). In some cases this rise subsides very quickly, or it may remain entirely absent; e. Cyanotic color, œdema, and perspiration are sometimes noticed on the paralyzed limbs (implication of the vaso-motor nerves). According to Nothnagel, some cases manifest symptoms of paralysis of the cervical sympathetic: unilateral redness and sweating of the face, narrowing of the palpebral fissure, dilatation of the pupil, and increase of the salivary, lachrymal, and nasal secretions.

As a rule, all the paralytic symptoms are most marked immediately after the attack. Very gradually some of them begin to subside. The first signs of returning power appear in the legs, next in the arms, but the latter rarely recover to the same extent as the former. The paralysis of the face often disappears almost entirely. Complete restoration of power is possible, but only when the motor tracts are merely compressed by the extravasation.

Associated movements constitute a peculiar phenomenon. While the patients are unable to perform voluntary movements of the paralyzed side, these occur involuntarily during laughing, crying, or sneezing. If the eye is blinded by a bright light, or the nasal mucous membrane or face is tickled, the patients not infrequently make reflex movements of repulsion with the paralyzed arm. If they are directed to make vigorous pressure with the healthy hand, involuntary pressure will be made with the other hand. Sometimes in attempting to move the paralyzed limbs, involuntary movements occur in the healthy limbs.

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In the form of hemorrhage under consideration, sensibility is unimpaired. Anæsthesia is frequent, immediately after the apoplectic attack, but is merely the result of pressure on remote parts and gradually disappears. If the sensory tracts are irritated, hyperæsthesia and paræsthesia are produced. Partial anæsthesia has also been described.

The reflex movements may be diminished or unaffected; when secondary degeneration sets in, they may be increased.

FIG. 115.

Position of the arm and leg in a man æt. 52 years, attacked by hemiplegia six months previously.

The paralyzed muscles maintain their normal volume for a long time; slight emaciation occurs at a late period as the result of disuse. The faradic and galvanic excitability of the nerves and muscles is retained, indeed it is said to be increased at times, during the first few days.

Trophic changes are sometimes noticed, such as abnormal development of hair on the paralyzed limbs, thickening and fissures of the nails, desquamation of the epidermis. The joints and sheaths of the tendons

are sometimes affected. The disease of the joints is shown by violent pains which generally begin one to three months after the paralysis. The affected limb is generally warmer and moister than the other. The shoulder joint is most frequently attacked, next the wrist, knee, elbow, and metacarpo-phalangeal joints. On autopsy, Charcot found injection and villous swelling of the synovial membrane, and in one case serous exudation. In seven cases, Hitzig noticed subluxation of the head of the humerus.

Gubler and Fournié noticed painful inflammatory swellings of the sheaths of the tendons which developed one to three weeks after the apoplectic seizure, and recovered in one to three months. Nodular thickenings of the peripheral nerves (neuritis nodosa) are sometimes observed.

The nerves of special sense are affected very slightly or not all. Occasional mention is made of disturbances of taste.

The vegetative functions may be unchanged. The most important phenomena in the stage of secondary changes are: Contractures of the paralyzed limbs, twitchings, increase of the tendon reflexes, which have been attributed to secondary degeneration of the pyramid tracts.

The contractures are more marked in the upper than in the lower limbs. The flexors are chiefly affected, so that the fingers, hand, and forearm are flexed. The arm is generally adducted against the thorax. In the leg, the extensors predominate over the peroneal muscles. The contractures diminish after prolonged rest, especially after sleep, and are increased on attempting to move the limbs. If they have lasted a long time, they give rise to changes in the joints which favor malposition of the limbs and increase the difficulty of movement.

The secondary changes also include the frequent impairment of the mental faculties. The patients are unusually irritable and whimsical, they laugh and cry without reason, become forgetful, and finally imbecile. According to Carpani, the intelligence is more impaired in right than in left hemiplegia.

The duration and course of this disease vary greatly; death sometimes occurs almost immediately; in other cases, life is prolonged ten to twenty years.

Cerebral hemorrhage presents a great tendency to relapses, after an interval varying from a few hours to months or even years.

If death does not occur during the apoplectic seizure, it is generally the result of increasing marasmus or intercurrent diseases (pneumonia, bronchitis, etc.).

IV. DIAGNOSIS.—The diagnosis is generally easy. We have previously referred to the possibility of mistaking the apoplectic seizure for drunkenness or opium poisoning. Apoplectiform attacks may also occur in circulatory disturbances and œdema of the brain, but these are not followed by permanent paralysis. It is not always easy to distinguish cerebral hemorrhage from embolism or thrombosis of the cerebral arteries. Special importance must be attached to the existence of valvular lesions as a source of emboli. Apoplectiform attacks are also observed in cerebro-spinal sclerosis, rarely in epilepsy and tabes dorsalis, but these attacks are not followed by permanent hemiplegia, and, in addition, other symptoms of the diseases in question are noticeable. Finally, such attacks also occur in progressive paralysis of the insane, but the mental disturbances and affection of speech predominate in this disease.

The localization of the hemorrhage is determined according to the

rules laid down in the preliminary remarks on the localization of cerebral diseases. The hemorrhage and paralysis are very rarely on the same side of the body. Such an occurrence can only be explained by the absence of decussation of the pyramid tracts.

Secondary degeneration of the pyramid tracts may be inferred from the presence of contractures, increased tendon reflexes, and twitchings in the paralyzed limbs.

V. PROGNOSIS.—The prognosis is always grave, since immediate death may ensue or irreparable injury is left over. The most dangerous hemorrhages are those into the pons and medulla, because these parts are the site of important centres. Rise of temperature and the development of trophic changes at the period of the apoplectic seizure have an especially unfavorable prognostic significance.

VI. TREATMENT.—Prophylactic measures must be adopted in those who have suffered from a previous attack of hemorrhage. Such individuals must avoid mental and bodily excitement, take non-irritating but nourishing food, avoid tea, coffee, and alcoholics, and secure a daily evacuation from the bowels.

During the apoplectic seizure, venesection is indicated if the pulse is hard, the carotids throb vigorously, the action of the heart ·excited, and the face congested. Under such circumstances the patients, after the venesection, not infrequently come out of the most profound coma and remain conscious. Venesection is contra-indicated if the pulse is small, the heart-sounds feeble, and the face pale. In the former event the head should be kept high, in the latter we may order irritants (injection of camphor, enema of vinegar, mustard draughts to calves, chest, etc.), and attempt to check the hemorrhage by injection of ergotin in the region of the forehead or ear, and the application of an ice-bag to the head on the side of the hemorrhage.

After the symptoms of the apoplectic attack have subsided, the diet should consist mainly of milk, soup, and soft-boiled eggs; wine may be administered cautiously to anæmic individuals. The bowels should be evacuated by a mild laxative. About the middle of the second week we may attempt to aid the absorption of the extravasation. Potassium iodide (gr. xij. t. i. d.), iodide of iron, and mercurial inunctions have been largely employed for this purpose without any very brilliant results.

In our opinion, the electrical current is a powerful therapeutic agent. We do not employ the galvanic current until all irritative symptoms have subsided, as a rule, not before the close of the sixth week. The current is applied as follows: a large electrode on the skull over the supposed site of hemorrhage, a small one immediately below and behind the angle of the lower jaw upon the upper cervical ganglion of the sympathetic, feeble current (six to eight elements), the cathode applied to the skull for three minutes, then the anode for the same period, three to four sittings a week.

The development of muscular contractures may be limited and almost prevented, if the position of the paralyzed limbs is changed several times daily, massage applied to the muscles, and the hand and fingers fastened to splints at night. The stabile galvanic current may be employed to relieve existing contractures.

The paralysis as such may be treated by faradism or by the labile galvanic current, the cathode on the paralyzed muscles, the anode on the back of the neck or over the focus in the brain.

Indifferent thermal springs and cold-water cures are also recommended, but should not be employed until the irritative symptoms have subsided.

The brain may also be the site of punctate or capillary hemorrhages which hardly exceed the dimensions of the head of a pin. They are most apt to occur in the cortex, sometimes singly, sometimes in very large numbers. They are sometimes so numerous and so closely aggregated that their effect is the same as that of a hemorrhage *en masse.* The surrounding parts of the brain are often softened and infiltrated with bloody serum.

On microscopical examination, a blood-vessel is found to form the centre of the capillary hemorrhages. If it is isolated by means of needles, the adventitious lymph sheath of the vessel is found dilated into an aneurism, and filled with red blood-globules. A rent can be detected at times in the walls of the vessel itself. Fatty degeneration and nuclear proliferation of the walls of the vessels are frequently found, but often are merely secondary to the hemorrhage.

In many places the adventitious lymph sheaths are also ruptured, so that the red blood-globules escape into the adjacent parenchyma, and extend more or less deeply between the nerve elements. Destruction of the nerve tissue often takes place, and granulo-fatty cells are then associated with the red blood-globules.

After punctate hemorrhages have lasted for some time, the red blood-globules degenerate, their pigment changes, and the foci assume a brownish, later an ochre-yellow color. Remains of the blood pigment are found in the adventitious lymph spaces, the perivascular lymph spaces, and the brain tissue (either free or inclosed in round cells in the form of yellow and brown granules, needles, or tablets). It has been supposed by some writers that the absorption of the blood and destroyed nerve tissue may give rise to the development of cystic cavities filled with serous fluid.

Punctate hemorrhages are most frequent in the course of infectious diseases and cachectic conditions (small-pox, splenic fever, pyæmia, puerperal fever, acute articular rheumatism, all diseases associated with dissolution of the blood, etc.).

The immediate causes of these hemorrhages are not always alike. In the majority of cases, it is probably the result of morbid permeability of the vessels and abnormally abundant diapedesis of red blood-globules, produced by morbid constitution of the blood and defective nutrition of the vessels; in other conditions—pyæmia and leukæmia—it is probably the result of embolic and thrombotic processes in the cerebral vessels. Finally, many authors regard fatty degeneration and fragility of the walls of the vessels as the cause of capillary cerebral hemorrhages.

5. *Embolism and Thrombosis of the Cerebral Arteries. Embolic and Thrombotic Cerebral Softening.*

(*Necrotic Softening of the Brain.*)

I. ETIOLOGY.—Stenosis or occlusion of cerebral arteries is produced either by obstructions to the circulation which have formed in situ (thrombi) or by those which have been carried into the vessels by the current of blood (emboli).

The most frequent causes of embolism are affections of the left heart, especially valvular lesions; inflammatory deposits or particles of the valvular tissue being carried off by the current of blood.

Among 297 cases of valvular disease of the left heart, embolism occurred 74 times, and in 15 cases affected the brain. Mitral lesions appear to be followed most frequently by cerebral embolism. The lesion may be the result of acute or chronic changes in the endocardium. In acute septic endocarditis, the emboli are generally very small, and obstruct only the capillaries; in chronic endocarditis, larger pieces are dislodged and occlude vessels of considerable size.

Emboli are sometimes secondary to cardiac thrombosis, which manifests a predilection for the left auricular appendix.

Next follow tumors (gumma, cancer), echinococci, abscesses, and aneurism of the heart muscle; these rupture into the cavities of the left heart or carry into them the clots which have formed in an aneurism.

In some cases, the emboli are derived from lesions of the aorta or carotid (ruptured atheromatous abscesses, coagula from aortic aneurisms, dilatation of the carotid and dislodgment of clots, tumors near the large vessels which have penetrated into their lumen, etc.).

Diseases in the distribution of the pulmonary veins may give rise to embolic changes in the brain. This may occur in ulcerating phthisical cavities, pulmonary abscess, cancer and gangrene, putrid bronchitis, the washing out of the pleural cavity after the operation for empyema (vide Vol. I., page 372).

Changes in the right heart cannot produce emboli of the aortic system (on account of the intervention of the narrow capillary system of the lungs) unless the foramen ovale is patent, so that emboli can pass directly from the right auricle into the left side of the heart.

Fat and pigment emboli are special forms of cerebral emboli, and usually affect the smaller vessels of the cerebral cortex.

Fat emboli generally follow fractures of the bones, and result from the entrance into the veins of the fat which has made its exit from the medullary cavities. H. Mueller also observed it after fatty degeneration of inflamed kidneys.

Pigment embolism is a sequel of grave intermittent fever. This leads to the development of black pigment in the blood which sometimes enters the vessels of the brain.

Cerebral thrombosis is generally the result of changes in the vessels. These sometimes develop after severe diseases (marantic thrombosis), such as pneumonia, typhoid fever, etc. It is probable that the cachectic condition gives rise to fatty degeneration of the endothelium of the intima, and that this affords opportunity for the accumulation of white blood-globules in such localities, and the formation of white thrombi.

Thrombi are more often the result of endarteritic changes. These sometimes develop as senile changes; sometimes they develop at an early period as the result of syphilis or alcoholism. They are sometimes so extensive as to produce almost complete occlusion of the arteries.

In syphilis, Baumgarten observed gummata in the adventitia and muscular coat of the cerebral arteries; these growths, particularly when associated with endarteritis obliterans, are apt to give rise to thrombosis.

In some cases, the thrombosis is the result of compression of the arteries, for example, by a tumor (compression thrombosis) or by inflammation in the vicinity, for example, meningitis (inflammatory thrombosis).

Thrombi in the cerebral arteries sometimes originate from thrombi of the carotid or vertebral arteries, but the continued deposit of purely thrombotic material finally causes them to extend to the cerebral vessels.

From the previous considerations it is evident that embolism and thrombosis will be associated with acute articular rheumatism, syphilis, alcoholism, cachexia, and old age. Embolism occurs more frequently in earlier years, thrombosis beyond the age of forty years.

II. ANATOMICAL CHANGES.—Cerebral embolism and thrombosis

affect most frequently the left middle cerebral artery, and, accordingly, the clinical history is found to possess a certain uniformity manifested by right hemiplegia and aphasia.

The predilection of emboli for the left middle cerebral artery is attributed to the fact that the left carotid is not given off from the aortic arch at such a sharp angle as the right carotid, and that the left middle cerebral artery is, in a certain measure, its direct terminal ramification.

If a cerebral artery is occluded by an embolus or thrombus, the effects will vary according as the occluded vessel may be supplied by adjacent and communicating vessels, or collateral circulation cannot be effected. In the former event, the disturbance of circulation subsides almost completely; in the latter, it gives rise directly to necrosis or to hemorrhagic infarction.

In the brain, the conditions vary according as the affected vessels belong to the circle of Willis and to the cortex, or to the arteries which pass from the circle of Willis into the basal portion of the cerebrum. In the former event, the effects of occlusion of the vessels are readily eliminated because the vessels communicate freely with one another. If the communicating branches in the circle of Willis are well developed, the brain may tolerate very marked lesions, as, for example, in Kussmaul's case, in which both subclavians and one carotid were occluded.

The conditions are entirely changed in occlusion of the arteries which originate from the circle of Willis and pass into the cerebrum at the base of the brain. These so-called terminal arteries do not communicate with adjacent vessels, so that their occlusion is necessarily followed by necrosis or hemorrhagic infarction.

Necrotic changes in the brain are known as softening (encephalomalacia); according to their appearance they are called white or gray, red or yellow softening. Cerebral softening is the result, not alone of embolism and thrombosis, but also of inflammation of the parenchyma of the brain (encephalitis). A distinction must be made, therefore, between necrotic and inflammatory encephalomalacia. Macroscopically, they are almost identical. Microscopically, the inflammatory form presents processes of proliferation in the cellular structures, but these may be concealed by the predominance of fatty elements, so that the discovery of an embolus or thrombus would be the sole factor to prove the necrotic character of the softening. In some cases, however, emboli or thrombi are gradually absorbed, and leave behind only their sequel, viz., encephalomalacia. Under such circumstances search must be made for embolic material in remote parts, or for endarteritic changes in the cerebral vessels (the latter would arouse the suspicion of previous thrombosis). Nevertheless the explanation of the case may remain impossible.

In white or gray softening of the brain, the affected part forms a white or gray, soft, occasionally almost diffluent mass, which often can be washed away by a stream of water, and leaves a cavity with jagged walls.

Microscopical examination of the necrotic tissues shows more or less advanced fatty degeneration of the nervous elements. The nerve fibres are the earliest to be affected, the ganglion cells are more resistant. Wengler has observed calcification of the ganglion cells. How long the nerve fibres may be deprived of blood without undergoing anatomical changes is not known with certainty, but there is no doubt that they will develop, at the latest, at the end of forty-eight hours.

The medullary sheath coagulates and breaks up into smaller and smaller

pieces, the axis cylinder also undergoes degeneration, next the cellular elements of the neuroglia and the vessels. Finally, nothing remains but fat granules, granulo-fatty cells, and empty, fatty remains of the blood-vessels.

The more the fatty degeneration progresses the more the gray or white color changes to yellow (yellow softening). This generally requires four to six weeks.

The absorbed fatty elements are sometimes replaced by a serous fluid, which is either clear or milky. This softening cyst may or may not be encapsulated. The cystic cavity is traversed occasionally by a network whose meshes contain fluid. The meshes consist in part of the remains of obliterated vessels, in part of newly formed bands of connective tissue. It is sometimes impossible to distinguish them from apoplectic cysts.

Red softening of the brain corresponds to the wedge-shaped hemorrhagic infarctions of other organs; in the brain, however, it is usually elongated and elliptical in shape. It is not a necessary result of embolism. The affected part appears red and soft, its periphery contains punctate hemorrhages. The microscope shows more or less changed red blood-globules and fatty detritus. The red blood-globules continue to degenerate and their pigment crystallizes. The part then assumes a brownish-red, chocolate, or ochre-yellow appearance, finally the character of yellow softening. We must avoid mistaking it for a hemorrhage (demonstration of an embolus).

The size of the softened spot sometimes exceeds that of an apple. A number of such spots are occasionally observed in various stages of development.

The emboli will possess infectious properties if they are derived from infectious foci, for example, putrid pulmonary processes, septic endocarditis, etc. They may then give rise to inflammation and abscess of the brain, indeed even to ichorous processes, if they are derived from gangrenous foci.

Little need be said concerning the changes in the vessels themselves. Emboli are often recognized as such by the mere fact that they are situated at the points of bifurcation of the arteries. The artery is not infrequently dilated into a spindle shape at the site of obstruction. In a case in which the embolus was derived from the lung, Boettcher detected pulmonary pigment in it. In thrombosis, the vessel is found thickened in places, calcareous, yellow, and hard, and gaping on transverse section. The thrombus may entirely occlude the vessel, it may be peripheral, or propagated from more remote parts. It may undergo absorption, canalization, fibrous metamorphosis, and calcification.

III. Symptoms.—Cerebral thrombosis often develops slowly, embolism suddenly. Thrombosis is preceded not infrequently by prodromata, such as headache, dizziness, nausea, vomiting, disturbances of sight and hearing, irritability, impairment of memory, temporary aphasia, paræsthesia, temporary paresis or paralysis. These are evidently the result of narrowing of the lumen of the vessel; but it appears as if the brain can accommodate itself to a certain diminution in the supply of blood and produces functional disturbances only when the minimum boundary is passed. This may occur as suddenly as in embolism.

The symptoms are then exactly like those of an apoplectic attack, and the terminations are also identical. It has been thought that embolism and thrombosis are associated more frequently with general or hemiplegic and monoplegic convulsions, but this does not justify a differential diagnosis between hemorrhage and embolism or thrombosis.

In many cases, the stage of apoplectic coma is followed, as in cerebral hemorrhage, by a stage of inflammatory reaction.

This is followed by the stage of focal symptoms, which vary according to the portion of the brain affected. Since embolic and thrombotic processes occur most frequently in the left middle cerebral artery, the symptoms usually consist of right hemiplegia and aphasia.

Embolic hemorrhages into the retina are sometimes observed as prodromal symptoms.

The secondary changes after embolism or thrombosis are the same as in cerebral hemorrhage (vide page 227).

The symptoms sometimes subside very rapidly, for example, if an embolus is broken up and the particles are driven into small, unimportant arteries. Relapses are not infrequent.

IV. Diagnosis.—The recognition of embolism and thrombosis of the cerebral arteries is one of the most difficult diagnostic problems. They may hardly ever be differentiated with certainty from cerebral hemorrhage. If the patient is young and has a demonstrable valvular lesion, if embolic phenomena have been noticed in other organs (hæmaturia, enlargement of the spleen, pulselessness in certain parts, etc.), if changes can be demonstrated in the aorta or carotids (aneurism) or embolic changes in the eyes, the diagnosis of embolism is more probable.

The differential diagnosis between cerebral hemorrhage and thrombosis is much more difficult. The latter is more probable if arteriosclerotic changes can be demonstrated in the peripheral arteries, if the middle cerebral artery is attacked, or if syphilis constitutes a probable etiological factor. But the diagnosis never can be made with more than a certain degree of probability.

The same factors must be taken into consideration in attempting to make a differential diagnosis between embolism and thrombosis of the cerebral arteries.

V. Prognosis.—The prognosis is also grave, because the disease may prove immediately dangerous to life; the foci of softening and the symptoms dependent thereon cannot be made to disappear. and relapses are frequent and cannot be prevented. Recovery is possible only in embolism of the cortical vessels.

VI. Treatment.—The treatment is similar to that of cerebral hemorrhage, except that stimulants, and not venesection, are indicated during the apoplectic seizure. In syphilitic cases, inunctions of mercurial ointment (℥ i. daily) and potassium iodide internally (gr. xij. t. i. d.).

6. Inflammation and Abscess of the Brain.

Cerebral Encephalitis.

I. Etiology.—Encephalitis and its termination in abscess are rare occurrences.

One of the most frequent causes is traumatism. As a matter of course, it occurs particularly after injuries to the skull. These include simple concussion, fractures and fissures, impaction of fragments of bone in the brain, entrance of foreign bodies.

The cerebral lesion is sometimes found at the site of injury, sometimes on the opposite side of the brain (contrecoup).

In some cases the inflammation is propagated from adjacent parts.

A prominent part is played by ear diseases. Tuberculosis or ordinary suppuration of the petrous portion of the temporal bone or the mastoid process is a not infrequent cause of encephalitis and cerebral abscess. In some cases the pus makes its way to the meninges and brain substance, and directly inflames them; or it creeps along the sheaths of the facial and acoustic nerves to the brain; or the blood-vessels and lymphatics convey the inflammation-producers to the brain, so that encephalitis or cerebral abscess (with or without thrombosis of the sinuses) is produced, despite the fact that the bones and nerve sheaths are intact. Diseases in other external cavities of the skull may also give rise to encephalitis, inasmuch as the inflammation is propagated directly to the brain.

This category includes tumors of the antrum of Highmore, of the nasopharyngeal cavity, and the orbits. True inflammations, for example, ozæna or orbital inflammations, are even more apt to extend to the brain. It is hardly necessary to say that ulcerating, tuberculous, and syphilitic changes in the skull are apt to attack the meninges and brain.

Embolic and metastatic processes sometimes play an important part in the etiology.

In the previous section we have called attention to the fact that cerebral emboli sometimes possess infectious and inflammation-producing properties (septic endocarditis, pulmonary abscess and gangrene, putrid bronchitis, bronchiectasis, empyema, suppurations at the periphery of the body and in the abdominal cavity, infectious diseases, and inflammations of the soft parts of the skull, etc.).

A fourth group of cases includes those in which a previous disease of the brain is followed by secondary inflammation. Thus, hemorrhages, necrotic softening, tumors, and parasites may set up inflammation in the surrounding parenchyma.

A series of cases remains in which no cause can be demonstrated (spontaneous cerebral abscess). The number of such cases diminishes with increasing care in post-mortem examinations, and many modern authors deny their occurence.

Men are attacked more frequently than women, because they are more subject to injuries.

II. ANATOMICAL CHANGES.—The inflammatory changes of encephalitis occur either as inflammatory softening or abscess. We distinguish red and yellow inflammatory softening. Necrotic and inflammatory softening may look exactly alike and often can be differentiated only by taking into consideration the previous history and other pathological appearances (embolus, thrombus, trauma).

In red softening, the affected spot exhibits a diminished consistence and a blood-red, in older cases brownish-red to chocolate color. At the centre, the color is uniform; at the periphery, we find patchy redness and punctate extravasations. On account of serous infiltration, the morbid focus occupies a greater space than that originally occupied by the inflamed portion of the brain. It projects, therefore, upon the cut surface or presses upon the entire hemisphere, as shown by flattening of the convolutions, dryness and anæmia of the surface of the brain. In some cases, the changes are confined chiefly to the immediate vicinity of the lesion, and are manifested by anæmia or inflammatory œdema, or both.

A distinction cannot be made between parenchymatous and interstitial encephalitis, since both parts of the parenchyma take part in the inflammation.

The neuroglia presents swelling of its cellular elements and the intercellular tissue, proliferation of the nuclei and development of myeloplaxes, cloudy swelling terminating in fatty degeneration. In the ganglion cells, swelling, nuclear

proliferation, vesicular transformation of the nuclei, cloudy swelling, finally fatty degeneration and destruction have been described. Destruction of the medullary sheaths of the nerve fibres, spindle-shaped distention, and later fatty degeneration of the axis cylinders, have been observed. Among the changes mentioned are extravasations of blood, so that free blood-globules are found singly or in groups between the nervous elements of the brain. The blood-vessels are dilated in places, their nuclei swollen and increased in number; here and there the adventitious lymph sheaths are dilated ampulla-like, and filled with extravasated red blood-globules. In some places, the vessels are surrounded by emigrated white blood-globules.

The transition from red to yellow softening takes place gradually, as the result of destruction of the extravasated red blood-globules and conversion of their pigment, but especially of the increasing fatty degeneration in the inflamed parts. As the final result of the former process, we find, as the remains of previous hemorrhages, hæmatoidin in the form of granules, needles, and tablets, partly free, partly inclosed in cells. Some of the ganglion cells are stuffed full of yellow pigment (so-called pigment degeneration). Calcification of the ganglion cells has also been observed. The main mass of the spot of softening is formed of granules of fat and granulo-fatty cells. The latter may result from fatty degeneration of the neuroglia cells, the nuclei of the vessels, and the ganglion cells, but in part they are fat-containing amœboid cells which transport the fat to the lymphatic and vascular channels. Hence the adventitious lymph sheaths of the blood-vessels are also filled with fat granules and granulo-fatty cells. The blood-vessels in part are obliterated.

If a stream of water is allowed to flow over the site of disease, the softened mass will be washed away, and leave an irregular cavity with jagged walls.

The fatty mass may be entirely absorbed and replaced by a firm, sclerotic cicatrix, which is pigmented particularly at its centre. As a matter of course, the nerve elements which have been destroyed do not undergo regeneration. If the cicatrix has formed in the cortex, it results in a more or less marked depression, over which the pia mater is thickened, and firmly adherent to the surface of the brain. This termination is similar to that of many hemorrhages, and indeed it is often impossible to determine whether the cicatrix is hemorrhagic or inflammatory in its origin. In other cases, the attempts at recovery lead to the formation of cysts. The fatty mass is gradually absorbed, and replaced by serous or cloudy fluid. The latter is encapsulated, the capsule being often traversed by connective-tissue septa, formed partly of new formed connective tissue, partly of obliterated vessels.

The number, size, and situation of the encephalitic foci are subject to great variation. In the majority of cases, there is only one focus, but under certain circumstances, for example, in pyæmia, there may be ten or twenty. They are usually so much larger the smaller their number. Pyæmic foci are often no larger than the head of a pin; solitary ones may attain the size of an apple or fist, or occupy almost an entire hemisphere. The cortex and basal ganglia are the most frequent sites of the disease.

Under certain circumstances, the inflammation terminates in the development of pus (cerebral abscess). Abscess of the brain may be diffuse or encapsulated.

In diffuse cerebral abscess, the collection of pus is directly in contact with the parenchyma of the brain. It exhibits a decided tendency to

spread more and more, and finally may lead to perforation into the ven-
tricles, towards the base of the brain (with secondary meningitis), or
upwards through the cortex. If there are openings in the skull, for
example, as the result of injury or ulcerative processes in the bones, the
pus may make its way externally. Diffuse cerebral abscess is often
irregularly elongated. It may be situated in the cortex or deep in the
substance of the brain.

In encapsulated cerebral abscess, the collection of pus is surrounded
by a fibrous capsule. This may attain a thickness of several millimetres,
and be very firm and resistant; it passes gradually into the surrounding
parenchyma. The encapsulation of an abscess does not appear to put
an end to its further growth. It may exceed the dimensions of an
apple.

There may be transitional forms between the two varieties of abscess,
i. e., only a part of the abscess may possess a cyst-wall.

The formation of pus in the brain may occur very rapidly, and in
some cases it has been found five days after injury. Encapsulation
occurs, on the average, from the seventh to twelfth weeks; in Lalle-
mand's case, it was found on the fifteenth day.

The pus in a cerebral abscess is generally acid, has a rancid odor,
and not infrequently presents a consistence like that of synovial fluid or
mucus. Coagula form on the addition of acetic acid. The pus may
become putrid if it communicates with the air through an opening in
the skull, or if it is produced by ichorous inflammation of surrounding
parts, or by ichorous emboli.

Fragments of bone or foreign bodies—the results of previous injury
to the skull—are sometimes found in the pus.

On microscopical examination the pus is found to consist, in great part, of pus-
corpuscles, which have been described as multinuclear (perhaps the result of pro-
longed maceration). In addition, drops and granules of fat, needles of margaric
acid, and, according to some writers, corpora amylacea.

The cyst-wall is composed of firm, fibrillated connective tissue, with spindle-
shaped cells scattered here and there. Rows of fat and pigment granules also are
found in places between the fibres. The abscess-wall is poor in blood-vessels. It
is probably produced chiefly by the adjacent neuroglia, a certain part being
played by emigrated white blood-globules which are converted into connective-
tissue cells.

Thickening, caseation, and calcification of the pus have been de-
scribed among the secondary changes in abscesses of the brain. It is
said that the pus may undergo gradual absorption, and the cyst-walls
become adherent to one another.

III. Symptoms.—Encephalitis and cerebral abscess may remain
latent during life, and be found accidentally in individuals who have
died from an intercurrent disease.

In other cases, an apoplectiform attack occurs, and proves more or
less rapidly fatal. The fatal event is occasionally preceded by a series
of apoplectiform attacks.

In a third series of cases, gradual exhaustion takes place, but the
causes remain latent during life. The most prominent symptoms are
anorexia, emaciation, sallow complexion, and increasing feebleness;
death results from exhaustion, and the autopsy reveals an abscess of the
brain.

In still other cases, peculiar febrile movement is noticeable (chills,
followed by high fever, and terminating in sweat). These attacks may

occur at such regular intervals as to rouse the suspicion of intermittent fever, particularly if the liver and spleen are enlarged. The fever is attended by emaciation and a cachectic appearance.

Two of my cases ran their course with gastro-enteritic and typhoid symptoms: gradual onset, continued fever, frequent vomiting, coated tongue, abdominal tenderness, obstinate diarrhœa, and ileo-cæcal gurgling, roseola, and slight enlargement of the spleen.

A suspicion of cerebral disease will only be aroused if diffuse or focal cerebral symptoms make their appearance. Even then the diagnosis will remain doubtful, unless an injury to the skull, ear diseases, or putrid processes in the organs of respiration point to a cerebral abscess.

The symptoms sometimes affect almost exclusively the mental sphere. The patients suffer from impairment of memory, gradually become imbecile or maniacal, etc.

In others, convulsive phenomena occupy the foreground; epileptiform attacks occur, at first rarely, then more and more frequently, and finally terminate in death.

Headache is one of the most constant of the diffuse symptoms. It may be constant, or present only at times, or increases in paroxysms. Its intensity varies from a feeling of dull pressure to pain of intolerable violence.

Some patients complain of a disagreeable sensation of a rolling or moving body within the skull; this is often felt only in certain positions.

Vomiting is sometimes very obstinate, in other cases it is almost entirely absent. Vertigo, insomnia, and irregularity of the pulse may also be mentioned among the diffuse cerebral symptoms.

Some patients complain of pain and paræsthesiæ in the limbs, in one limb, or in hemiplegic or paraplegic distribution.

The changes in the fundus of the eye constitute, to a certain extent, the transition from the diffuse to the focal symptoms. The fundus often, though not constantly, presents the signs of choked disk, retinitis, and neuro-retinitis, which terminate in optic atrophy, after they have lasted a long time. The changes are sometimes more marked in one eye (on the side of the abscess). Retinal hemorrhages have also been observed.

The focal symptoms include paralyses, spasms, and contractures, the distribution of which depends upon the situation of the inflammatory focus in the brain.

Toynbee showed that diseases of the external auditory canal are followed most frequently by abscess of the cerebellum, those of the tympanic cavity by abscess of the cerebrum, those of the labyrinth by abscess of the medulla oblongata.

Among the unusual symptoms is escape of the pus externally. If the abscess ruptures into the ventricles, death generally occurs very rapidly, preceded by epileptiform convulsions. Perforation beneath or through the pia mater also gives rise to general convulsions, apoplectiform seizures, meningitic symptoms, and speedy death.

The duration and course of encephalitis and cerebral abscess vary extremely. In Haertlin's case, the abscess ran an entirely latent course for twenty-six years. In other cases, death occurs in a few hours or days.

A prolonged latent course often is interrupted suddenly by violent symptoms which subside, perhaps reappear. etc.

IV. DIAGNOSIS.—It is evident from the previous remarks that the detection of encephalitis and cerebral abscess is often impossible. The diagnosis includes the recognition of the presence of an abscess and its location. The first point is determined by the etiology, the second is based on the principles laid down in the preliminary diagnostic remarks.

V. PROGNOSIS.—The prognosis is always grave, almost always fatal. After suppuration has taken place, recovery cannot be looked for unless the pus is removed artificially by operation.

VI. TREATMENT.—Prophylactic measures (concerning which we refer the reader to special treatises), must be adopted in injuries to the skull and diseases of the auditory apparatus. If there is reason to assume the existence of encephalitis, we should order an ice-bag to the head, local abstraction of blood, inunctions of mercurial ointment, and mild laxatives, or, if the bowels are regular, potassium iodide. After pus has formed, the treatment consists of trephining and evacuation of the pus, if the abscess can be localized. Otherwise, purely symptomatic treatment.

APPENDIX.

a. Congenital encephalitis. Virchow has shown that encephalitis may sometimes be congenital. He observed it in still-born children and in new-born, whose mothers had suffered from small-pox or syphilis. Birch-Hirschfeld observed it in children suffering from jaundice and suppuration of the umbilicus. But not every discoloration of the brain and accumulation of granulo-fatty cells may be regarded as an inflammatory product, since Jastrowitz has shown that the development of the medullary sheaths in the fœtal brain is attended with the formation of granulo-fatty cells. While the diffuse occurrence of granulo-fatty cells is physiological, their aggregation in foci is the result of an inflammatory process; in addition, proliferation of round cells and hemorrhages are often present.

b. Acute cerebral, atrophic infantile paralysis. Acute infantile polio-encephalitis. This disease generally attacks children between the ages of one to three years. As a rule, no cause can be demonstrated; an infectious influence appears very plausible.

As a rule, the disease begins acute and suddenly. The children are attacked with fever, vomit, become stupid, and are seized with epileptiform spasms. They generally recover consciousness in one or two days; hemiplegia is then noticeable. In the majority of cases, the limbs alone are affected, but more rarely the facial or other cerebral nerves are paralyzed. Monoplegia is sometimes observed.

The paralysis occasionally disappears in certain parts. The permanently paralyzed muscles soon undergo atrophy, and the growth of the paralyzed limbs is retarded. The panniculus adiposus is increased in thickness, the skin is cold, and has a blue, marbled appearance. Sensibility is generally unchanged, the tendon reflexes exaggerated. There is sometimes a marked tendency to profuse perspiration.

The paralyzed muscles gradually become rigid, and muscular contractures may develop. Motor irritative symptoms set in not infrequently in the form of hemichorea, hemiathetosis, or associated movements. At a subsequent period, the children often suffer from epilepsy or become idiotic.

The clinical history is very like that of acute poliomyelitis (vide page 118), and probably it is the result of corresponding anatomical lesions. There is no doubt that acute polioencephalitis is an acute inflammation of the motor cortical centres.

Autopsies have been made hitherto in old cases alone; the lesions found were atrophy of the cortex, the formation of cysts, thickening and adhesions of the pia mater. In rare cases, the disease is the result, perhaps, of cortical hemorrhage or embolism.

The prognosis and treatment are the same as in acute poliomyelitis (vide page 120).

7. *Tumors within the Cranial Cavity.*

I. ETIOLOGY.—Intracranial tumors may grow from the brain substance, the meninges, or the bones of the skull. Extracranial tumors sometimes proliferate into the cranial cavity through openings in the skull. This may happen in the case of orbital tumors which pass through the optic foramen into the cranial cavity; or tumors of the nasal cavity, the antrum of Highmore, the spheno-palatine fossa, or the petrous portion of the temporal bone, after eroding the bones which cover them, may also proliferate into the brain. On the other hand, tumors within the cranial cavity sometimes perforate the bones of the skull and grow outside.

Very little, if anything, is known concerning the real cause of development of intracranial tumors. We are satisfied of the influence exerted by traumatism, although this was strenuously denied by Cohnheim.

Intracranial tumors may be the result of chronic infectious diseases, particularly tuberculosis and syphilis (solitary tubercles and gummata), but these will not be considered here.

Metastatic growths are sometimes found, following neoplasms (cancer and sarcoma) in other organs.

Some writers attach importance to hereditary influences. The development of intracranial tumors is also attributed to excessive mental strain, excesses in Baccho et Venere, and exposure to the sun.

They are more frequent in men than in women, probably from the greater liability of the former to suffer injuries, to indulge in alcoholic excesses, and to the greater demands upon their mental activity.

Cerebral tumors occur at every age (Hasse found a cancer of the brain in a new-born babe). Tubercles are more common in children (usually beyond the age of 2 or 3 years), while cancer is observed more frequently at an advanced age.

II. ANATOMICAL CHANGES.—Two factors must be taken into consideration—the structure of the tumor, and its effects upon the brain.

The following are the varieties of tumors (in their order of frequency) found in the cranial cavity: glioma, sarcoma, psammoma, myxoma, carcinoma, melanoma, cholesteatoma. Very rare are: papilloma, fibroma, lipoma, enchondroma, osteoma, dermoid cysts, cysts, and angioma.

Allied to tumors are the cases of heterotopia of the gray matter. This term is applied to an accumulation of gray matter in abnormal positions, occasionally in the form of small nodules, which sometimes project like a tumor into the ventricles.

Among the tumors should also be included hyperplasia of the pineal gland and struma pituitaria.

Gliomata generally occur singly and start from the white matter; if they extend to the surface of the brain, they form no adhesions to the meninges; they develop with striking frequency as the result of injury, grow very slowly, and sometimes attain almost the size of a fist. Their color often differs very little from that of normal gray matter, so that small tumors are readily overlooked; in other-

cases, they have a more or less bright red color, according to their vascularity. The latter is often very great, thus affording the possibility of rapid changes in size, and clinically of rapidly alternating symptoms of irritation and paralysis; furthermore, of extravasations of blood. The latter may resemble ordinary cerebral hemorrhages, and the diagnosis often cannot be made until tumor masses are discovered at the peripheral zones of the hemorrhage. Virchow also states that fatty degeneration of a glioma is apt to be mistaken for yellow softening of the brain. Small gliomata may simulate sclerotic foci, but the latter are generally multiple. These tumors start from the neuroglia, so that they are indistinctly separated from the adjacent parenchyma of the brain. There may be transitions between glioma and myxoma or sarcoma (myxo-glioma and sarcoglioma). The former are soft and gelatinous on account of the mucoid character of the basement substance ; the latter are rich in cells, tougher and firmer.

Sarcoma of the brain is generally primary, more rarely metastatic, or the expression of general sarcomatosis. Melanosarcoma has a special tendency to the formation of metastatic growths in the cranial cavity.

As a general thing, only one sarcoma is found in the cranial cavity, more rarely there are several. They may start from the meninges or substance of the brain. Sarcoma of the dura mater is relatively frequent at the base of the skull, and is especially apt to grow from the sella turcica and petrous portion of the temporal bone.

Sarcomata of the substance of the brain generally possess a vascular membrane around them, so that they can readily be peeled out of the brain. The tumors may be firm or soft, whitish in color, or vascular. In the latter event they exhibit a tendency to hemorrhage. They are distinguished from gliomata by their more rapid growth. The cells of sarcoma are very delicate, so that in fresh, teased preparations we find chiefly free nuclei.

Psammoma is characterized by the occurrence of chalky concretions in its tissues. The latter are often visible to the naked eye as yellowish granules, grate under the knife, and are composed of laminated masses of carbonate of lime. The chief mass of the tumor is fibrous. It exhibits a predilection for the dura mater, particularly of the convexity of the brain. The tumors are generally single, rarely multiple, and do not often exceed the size of a walnut. They are sometimes found in the brain substance itself.

Carcinoma rarely develops in the cranial cavity. In the majority of cases the tumor is primary and usually single. Multiple cancers are sometimes situated in symmetrical localities. Cruveilhier counted over one hundred tumors in one case. Their shape is round, nodular, lobulated, more rarely they are diffusely infiltrated. They may start from the dura mater, pia mater, or substance of the brain, and occasionally grow into the cranial cavity from the outside. On the other hand, tumors which start from the outer surface of the dura mater sometimes perforate the skull and appear beneath the scalp. They sometimes attain the size of a fist. Medullary cancer is frequent, scirrhus is rare. According to Rindfleisch, all cancers of the brain start from the pia mater, although their connection with the latter may become indistinct. They often possess a capsular membrane. Fatty degeneration, caseation, calcification, and ossification have been described as secondary changes.

Myxomata and melanomata are rare growths ; the latter develop from the pigment cells of the pia mater.

Cholesteatomata are firm, epithelioidal tumors, with a mother-of-pearl gloss, and sometimes attain the size of a walnut.

Intracranial tumors may injure the brain temporarily and permanently—temporarily by changes in volume as the result of varying vascularity, permanently by increasing growth, compression, and irritation. If the tumor starts from the bones or meninges, the surface of the brain beneath is flattened, the gyri and sulci are effaced, the surface often dry and unusually anæmic. Tumors at the base of the skull often exert pressure on the cerebral nerves and cause them to undergo atrophy.

In tumors of the substance of the brain, the affected side often appears more prominent; when the dura mater is thrown back, the surface is flattened, dry, and anæmic. The falx cerebri and the brain itself are sometimes pushed towards the opposite side (vide Fig. 116).

The vicinity of the tumor may undergo important changes: suppuration, softening, hemorrhage, and anæmia. New growths are followed occasionally by meningitic symptoms, more frequently by hydrocephalic changes as the result of pressure on the vena Galeni.

III. SYMPTOMS.—Intracranial tumors sometimes run an entirely latent course, and are discovered accidentally at the autopsy. This may be true of relatively large neoplasms, while, on the other hand, very small ones may produce the most violent symptoms. This depends upon the localization and rapidity of development.

Among the diffuse symptoms of intracranial tumors, headache occu-
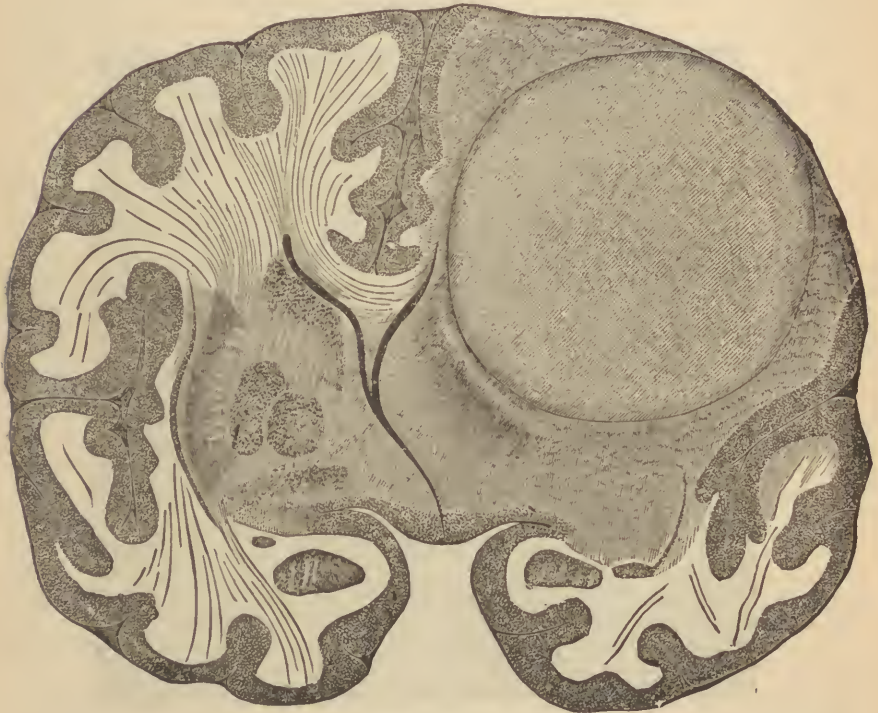
Cerebral tumor, producing displacement. After Obernier.

pies a prominent part, and it is so constant that its absence contra-indicates the diagnosis of tumor in a doubtful case. The pain may be constant or intermittent, occasionally it is felt chiefly at night. It is provoked or intensified by bodily and mental excitement, and excesses in Baccho et Venere. Its intensity may become so extreme that the patients grow wild with pain. In other cases it consists merely of a feeling of dull pressure within the skull. The patients are often unable to localize the pain accurately. As a general thing, its distribution does not throw much light on the location of the tumor. But if the pain is referred constantly to the occiput and nape of the neck, the tumor may be assumed to be situated in the posterior fossa (occipital lobes, more fre-

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quently the cerebellum). The pain is probably the result of dragging upon the meninges, which are rich in nerve fibres.

The skull is sometimes very tender on percussion. If the tenderness is confined to a circumscribed spot, it may be assumed to be connected with the situation of the tumor; but this is a rare occurrence.

Very many patients complain of vertigo. It is sometimes so constant and severe that the patients are unable to move about unsupported. They often exhibit a tendency to fall in a definite direction, backwards or forwards, to the right or left. This symptom is apt to be especially well marked in tumors of the posterior fossa.

Psychical disturbances are of frequent occurrence. The patients be-

Fig. 117.

Choked disk in cerebral tumor. After Magnus.

come moody, irritable, apathetic, somnolent, the memory is impaired; delirium, maniacal attacks and well-marked psychopathies set in, and sometimes obscure the real cause of the disease. The mental disturbances also present remissions and exacerbations.

Conditions of somnolence and coma occur with remarkable frequency; they may last hours, days, or even weeks.

Apoplectiform and epileptiform attacks are by no means infrequent. The former may be the result of hemorrhages into the tumor or its vicinity, or, like the epileptiform seizures, they are the result of irritation produced by sudden increase of cerebral pressure. Hence they are followed, according to circumstances, by permanent or transitory

paralyses. The epileptiform spasms are general, unilateral, confined to one limb or to groups of muscles. In some cases, consciousness is retained. Epileptiform twitchings in the distribution of a single nerve, or recurring spasms in one limb alone, must arouse the suspicion of irritation of parts of the motor region of the cortex.

Choreiform movements sometimes appear in individual limbs. This symptom has been attributed to a lesion of the posterior part of the internal capsule, but it has also been observed in diseases of other parts, for example, the pons Varolii.

Many patients complain of vague, peripheral, rheumatoid pains, and occasionally of intolerable itching of the skin.

Changes in the fundus of the eye and in vision constitute some of the most important diagnostic symptoms. The most important is the so-called choked disk (papillitis), which is recognized by swelling of the optic nerve, distention of the retinal veins, and narrowing of the arteries (vide Fig. 117). Reich found that among eighty-eight cases of tumor of the brain, choked disk was present in 95.4%. Sight is impaired in many cases; amblyopia is produced and even complete amaurosis. It must be remembered, however, that the most marked evidences of choked disk may be present, although vision remains intact.

As a rule, choked disk is bilateral, although one eye may be more affected than the other. The lesion is the result of the increased intracranial pressure, in consequence of which cerebro-spinal fluid is forced from the subarachnoid space between the external and internal optic sheaths (intervaginal space). The dropsy of the optic sheath compresses the retinal veins, produces venous congestion, and, at the same time, swelling of the optic papilla. The changes in question subside at times, so that mere traces remain. In other cases, they are followed by retinitis, neuro-retinitis, and atrophy of the optic nerve, but the latter may also be the result of direct pressure of the tumor on the chiasm or optic tract.

Choked disk is also found in hydrocephalus, abscess, and menigitis, in short, in increased intracranial pressure from whatever cause, but it is most constant and marked in cerebral tumors.

Tumors of the brain are sometimes attended by retinal hemorrhages and by yellow patches, as in Bright's disease.

With regard to narrowing of the field of vision and disturbances of the color sense, we refer the reader to text-books on ophthalmology. Some patients complain of subjective sensations of light. Sudden blindness (apoplectiform amaurosis) occurs occasionally; it may be unilateral or bilateral, disappear and return. The ophthalmoscope shows no striking changes, and the symptom is probably the result of temporary increase of intracranial pressure.

Disturbances of the olfactory and acoustic nerves are sometimes observed, as the result of direct pressure on the nerve trunks, of neuritis, or of increased intracranial pressure, which is propagated to the sheaths of the nerves as in choked disk.

The general nutrition may be undisturbed, indeed there is sometimes an unusual development of the panniculus adiposus. Other patients are very pale and present an appearance like those suffering from Bright's disease. Cachexia develops rapidly if the tumor is cancerous.

The appetite is often lost, but in two cases we noticed insatiable boulimia. Increased thirst is often noticeable, particularly if the patients suffer much from vomiting. The urine may contain sugar if the tumor is situated in the floor of the fourth ventricle; Schultze found inosit in two cases of this kind. The bowels are usually constipated; involuntary evacuations may occur during comatose and apoplectiform

conditions. Sexual desire is increased in some patients, in others it is lost. During comatose and somnolent conditions, the patients often play with their genitals, but some writers regard these movements as purely mechanical and automatic.

Irregularity in breathing, Cheyne-Stokes respiration, and retardation of the pulse are frequent symptoms, which occur solely or chiefly during comatose conditions. Fever sometimes appears as a terminal symptom.

The focal symptoms consist of irritative and paralytic phenomena (spasms and contractures, pareses and paralyses). The previous remarks on the local diagnosis of brain diseases hold good here. Special importance attaches to paralyses of cerebral nerves of a peripheral character, because they are generally the result of compression by tumors at the base of the brain. The paralyses retain the peripheral character even when the nuclei of the nerves are destroyed by the tumor. Paralyses of central character only develop when the nerve-tracts are injured on the central aspect of the nuclei.

The sensory nerves quite often present the signs of anæsthesia dolorosa, because the tumor compresses the nerve and prevents conduction from the periphery, while it irritates the central end, and thus produces pain which is referred to the periphery.

The duration of intracranial tumors varies; calculations are not reliable, because it is very difficult to determine the beginning of the disease. Some years ago I saw a case of glioma of the brain which had lasted at least twelve years (v. Graefe had made the diagnosis from the presence of choked disk twelve years before death). Andral reports a case which lasted fifteen years. But, as a rule, they run their course in one to two years, sometimes in a shorter time.

Death may occur suddenly in an apoplectiform or epileptiform seizure, after a longer or shorter period of coma, or after bodily or mental exertion, for example, coitus or a drinking bout. The fatal termination is sometimes preceded by meningitic symptoms. If the tumor perforates externally, the scalp grows prominent and red, the hairs fall out, the tumor often exhibits a sort of fluctuation and respiratory movements; dulness, vertigo, and convulsions are produced if the tumor is pushed back. In rare cases, the patient is relieved by this manipulation. The edges of the opening in the skull are sometimes everted, and furnish a sensation of crepitation. If the integument is also perforated, the tumor ulcerates and often becomes gangrenous.

IV. DIAGNOSIS.—The recognition of cerebral tumors is occasionally impossible. Choked disk is the most important symptom. The disease may be mistaken for: *a. Cerebral hemorrhage ;* the advanced age and sudden onset of the symptoms deserve special consideration. *b. Cerebral thrombosis and embolism ;* the diagnosis of embolism is favored by the existence of valvular disease of the heart; in thrombosis the etiology must be considered; the latter is of still greater importance in the differential diagnosis from *c. cerebral abscess* (traumatism, suppuration of bone). *d.* Patients who are found in a comatose condition may arouse the suspicion of *uræmia,* but this will be decided by examination of the urine. *e.* The differentiation from *epilepsy* and *psychopathy,* without material changes in the brain, is hardly possible if the ophthalmoscopic appearances are negative. *f. Hysteria* is sometimes simulated if the patients present only ill-defined focal symptoms. *g.* In a case which I saw in consultation, and in which choked disk alone pointed to the real diagnosis (which was confirmed on autopsy), the pallor of the patient

led the attending physician to make the diagnosis of *progressive pernicious anæmia.*

V. PROGNOSIS.—The prognosis is unfavorable. The disease generally progresses uninterruptedly to a fatal termination.

VI. TREATMENT.—The treatment is purely symptomatic. The patient should eat easily digested, nutritious food, secure a daily evacuation from the bowels, and avoid bodily and mental exertion. Coitus and alcoholic stimulants must be interdicted. Morphine may be given subcutaneously to relieve headache and vomiting, potassium bromide (℥ iiss.–v. at a dose) may be used in conditions of great excitement.

Godlee recently enucleated a glioma which was situated below the right anterior central convolution. After temporary improvement, the patient died in the fourth week from meningitis.

8. *Animal Parasites in the Cranial Cavity.*

Two animal parasites (cysticercus and echinococcus) may be present in the cranial cavity; the former is found more frequently than the latter.

FIG. 118.   FIG. 119.
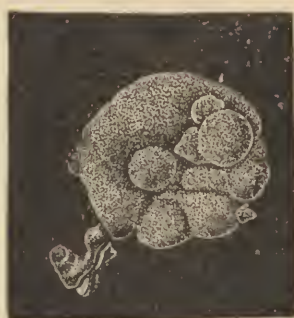
Cysticercus racemosus. After Marchand.

a. Cysticercus cellulosæ. Cysticerci of the brain are not very rare. They may start from the meninges, from the parenchyma of the brain (generally gray matter), the subarachnoid space, or they may be free in the ventricles. Those that start in the meninges grow generally from the pia mater, rarely from the inner surface of the dura mater. They press upon the surface of the brain, and bore depressions into it. In many cases there is only one vesicle, in others there may be several hundred, so that the surface of the brain and the meninges are strewn with them. The larger ones attain the size of a walnut, in exceptional cases that of a hen's egg or apple. The wall of the cyst is usually surrounded by a fibrous capsule, with onion-like lamellæ. The contents of the cyst are fluid. At a certain part, which is generally recognizable externally by a depression and thickening, is situated a prominence to which the neck and head of the worm are attached. The head is darkly pigmented at its apex, and the microscope shows a row of hooks and four suction disks. The cysticerci are said to live three to six years. After death of the parasite, the cysts undergo thickening, caseation, and calcification, and might be mistaken for degenerated tubercles and gummata, were it not for the persistence of the hooks.

The immediate vicinity of the cysticerci is sometimes entirely unchanged. In other cases, we notice interstitial proliferation of connective tissue or anæmia, hyperæmia, punctate extravasations, softening, inflammation, and suppuration. Hydrocephalus is not infrequent.

Cysticercus racemosus exhibits a special mode of growth. The cyst forms inversions, acquires daughter vesicles, like echinococci, and may form a multilocular structure (vide Figs. 118 and 119). This peculiar mode of development is attributed to the favorable conditions for growth, such as are found particularly in the subarachnoid cavity at the base of the brain.

Cysticercus racemosus should not be mistaken for free cysticerci which have become adherent to the wall of a vessel (vide Fig. 120). This variety is also relatively frequent at the base of the brain.

The paths followed by the parasites in their passage from the gastro-intestinal tract to the brain are not known with certainty. The parasites are more frequent in men, and develop, as a rule, beyond the age of twenty years.

The cysticerci sometimes run an entirely latent course, in others they give rise to well-marked insanity. In still others, the symptoms of a cerebral tumor become noticeable, especially irritative phenomena: headache, vertigo, vomiting, neuralgia, epileptiform attacks; paretic or paralytic symptoms are rare.

The diagnosis is often uncertain. Cerebral symptoms may be attributed, with great probability, to intracranial cysticerci only when the parasites are also found in the subcutaneous cellular tissue, the intermuscular connective tissue, or the fundus of the eye.

The prognosis is unfavorable, since there is no means at our command of killing the parasite. The treatment is purely symptomatic.

b. Echinococci are generally found in the brain as single vesicles, more rarely they are multiple. They vary in size, but occasionally attain the dimensions of a cerebral hemisphere. Morgan described one vesicle as large as a cocoanut, and weighing eighteen ounces. The vesicles sometimes perforate the skull and discharge their contents externally; spontaneous recovery may occur in this manner. Perforation through the nose and ear has also been described.

FIG. 120.

Free cysticercus upon an artery at the base of the brain. Natural size. After Heller.

The cysts often contain daughter vesicles, but not infrequently they are sterile and contain no scolices. The cyst-wall is generally surrounded by a fibrous capsule. The adjacent parenchyma may be anæmic or hyperæmic, contains extravasations, or in rare cases is entirely normal.

Intracranial echinococci often develop in young individuals. The symptoms are those of a brain tumor with predominant irritative symptoms, particularly epileptiform convulsions. Exophthalmus and œdema of the lids may be produced when perforation is about to occur. In Westphal's case, more than ninety vesicles were spontaneously discharged externally. Perforation externally may be followed by cure or merely by temporary relief. Death is sometimes apoplectiform.

The diagnosis is often impossible. The prognosis is very grave, since treatment could only prove successful by surgical removal of the parasites.

9. *Aneurism of the Cerebral Arteries.*

1. The causes of the development of aneurisms of the cerebral arteries are often unknown. Among those mentioned are: injuries to the head, abuse of alcohol, and syphilis. The remainder of the vascular system is often intact, in other cases aortic aneurism is also present. A combination with valvular endocarditis has often been noticed, and some English writers believe that cerebral aneurisms are, in part, of embolic origin.

They are more frequent in men than in women, and not infrequently occur at an early age. Among fifty-seven cases collected by Lebert,

         13 (24%) occurred from   0–19 years.
         22 (38%)   "   "   20–39   "
         22 (38%)   "   "   40–60   "

Among seventy-nine cases collected by Coats, forty-two occurred between the ages of ten to forty years, thirty-seven from the age of forty to eighty years.

2. In almost all cases, we have to deal with true aneurisms, *i. e.*, all the coats of the vessel are involved. Their size varies from that of a pea to that of a walnut, or in rare cases even larger. They are sometimes filled more or less completely with old clots. The adjacent nerves often suffer compression and atrophy, the parenchyma of the brain is softened. In three-fourths of the cases, the

aneurism ruptures, giving rise to profuse meningeal (particularly subarachnoid) hemorrhage, occasionally to destruction of the adjacent substance of the brain.

The aneurisms are found most frequently in the circle of Willis and its peripheral branches, more rarely in the internal carotid or vertebrals. Gairdner described an aneurism of the middle meningeal artery.

The most frequently affected vessel is the middle cerebral artery, next the basilar. Left-sided aneurisms are more frequent than those on the right side, occasionally they are multiple.

3. Aneurisms of the cerebral arteries may be entirely latent during life. In some cases, insanity or epilepsy develops; in others, there are diffuse and focal cerebral symptoms: headache, vertigo, vomiting, neuralgia, blindness, optic atrophy, twitchings, and paralyses in certain nerve tracts. Of course, these symptoms depend on the situation of the aneurism. Paralysis of the motor oculi nerve is especially frequent. Rupture of the aneurism is followed by an apoplectic attack, which, as a rule, is very rapidly fatal. Some patients complain of a subjective auditory sensation of roaring, and vascular murmurs have sometimes been heard on auscultation of the skull.

The following case may serve as an illustration of the history of the disease. A servant, æt. 23 years, who had suffered several weeks from vertigo, nausea, rush of blood to the head, and insomnia, noticed for several days a weakness of the right limbs. On admission to the hospital, there was paresis of all the branches of the facial nerve, and of the right arm and leg; in addition, paralysis of the left motor oculi nerve. Hence a diagnosis of disease of the left cerebral peduncle. A few days later, the patient had an apoplectic attack, and died thirty-six hours later. It was found that an aneurism of the left posterior cerebral artery, which had pressed on the cerebral peduncle, had undergone rupture.

4. The diagnosis is very difficult, almost always uncertain. The prognosis is unfavorable on account of the tendency to rupture; treatment purely symptomatic.

10. *Hydrocephalus.*

Hydrocephalus is the term applied to a collection of serous transudation, either between the meninges or into the cerebral ventricles. The former is known as external hydrocephalus, the latter as internal hydrocephalus.

In external hydrocephalus, the fluid may be situated in the subdural cavity, or in the subarachinoid meshwork. It is now believed that the latter variety is much more frequent than the former.

Intermeningeal and ventricular hydrocephalus are not entirely distinct, since, as is well known, the subarachnoid space communicates with the cavity of the ventricles. Moreover, observation teaches that effusions may be present at the same time in both localities.

Hydrocephalus may be diffused or circumscribed. The former is much more common than the latter. In the second variety, the accumulation of fluid is confined to circumscribed parts of various cavities. In this manner, cystic spaces develop between the meninges, or certain portions of the ventricles are dilated and filled with fluid.

In order that a large amount of fluid may enter the skull, the bones must yield to the pressure of the fluid and undergo distention, or the brain substance must be compressed. Distention of the skull can only occur during childhood. It is apt to be especially marked before the closure of the sutures and fontanelles. In adults, the fluid exercises its chief effect in producing compression of the brain, though not infrequently the cranial bones also present changes. They are unusually thin, the diploë is remarkably atrophied, in places the external and internal tables are in contact with one another, and the inner surface of the latter is often rough and uneven.

Hydrocephalus may be acute or chronic. In the former the fluid sometimes accumulates in a few hours, to an extent incompatible with

life; in the latter the disease may last months and years. Finally, hydrocephalus may be acquired or congenital.

a. Acquired Hydrocephalus.

I. ETIOLOGY.—The causes of acquired hydrocephalus include all those conditions which give rise to œdema in other parts. It occurs also in inflammations as inflammatory œdema.

Hydrocephalus is an almost constant element of purulent and tubercular meningitis. It may also occur in chronic inflammations. In such cases, ventricular hydrocephalus is generally associated with changes in the choroid plexus and the ependyma. Hydrocephalus occasionally develops after, not during the meningitis.

In some cases this condition is the result of stasis of blood.

The causes of stasis may be situated inside or outside of the skull. They include: tumors and inflammatory products which compress the vena Galeni or the sinus rectus of the dura mater; cardiac and pulmonary affections, and diseases in the mediastinum and neck, when associated with obstruction to the current of blood in the internal jugular vein; whooping-cough, in which the violent spasms of cough give rise to stasis.

In some cases hydrocephalus depends on cachectic and hydræmic conditions (cancer, phthisis, Bright's disease, etc.).

A special form is that known as hydrocephalus ex vacuo, which is found chiefly in old people who suffer from senile atrophy of the brain.

In a considerable number of cases, no causes can be found, or, at least, their significance remains doubtful. Alcoholism, syphilis, mental strain, and the effects of high temperature on the head have been regarded as causes of the disease.

There is no doubt that rachitis is associated with remarkable frequency with hydrocephalus.

The age of childhood appears to present a predisposition to the disease.

II. ANATOMICAL CHANGES.—Hydrocephalus is manifested in many cases, particularly in children, by the peculiar shape of the head. The head is unusually large, the frontal bones ascend straight upwards and forwards, the orbital cavities appear flattened, the fontanelles are unusually large, so that the large one extends to the region of the glabella, and the bones are separated by abnormally wide sutures. If ossification of the sutures takes place, it often occurs from the development of numerous supernumerary bones in the sutures. The hair of the scalp is generally thin, dry, and scanty. Dilated and sinuous veins are often noticeable on the forehead, temples, and vertex. The face appears unusually small, and is often triangular, with the base directed upwards, the apex at the chin.

The bones of the skull are sometimes remarkably thin, so that they appear transparent when a light is held behind them.

The remainder of the skeleton often presents evidences of rachitis; enlargement of the epiphyses, deformities in the limbs and thorax, irregular development and poor formation of the teeth.

In adults, and in children past the age of seven, the changes in the skull remain absent, indeed, the skull is sometimes very small, for example, in cretins.

In external hydrocephalus, the surface of the brain is often flattened and anæmic. In internal hydrocephalus, the brain is pushed outwards, but the convexity also appears flattened and anæmic. The ventricles are found to be distended and filled with serous, more rarely flocculent, cloudy, or bloody fluid. The flocculi are generally composed of desquamated epithelium cells, occasionally also of softened portions of the parenchyma. The walls of the ventricles, particularly the basal ganglia, appear flattened. They are occasionally softened and somewhat deliquescent, the result in great part of post-mortem changes (so-called white or hydrocephalic softening). The brain is sometimes so distended and thin that it resembles a vesicle. It may undergo rupture, almost always upon the convexity. The ependyma of the ventricle is often thickened and granular, particularly in chronic hydrocephalus, and its blood-vessels are sometimes dilated and sinuous. The choroid plexuses may present thickenings, sometimes cystic formations.

These changes appear earliest, and are the most marked in the lateral ventricles, next in the third, least frequently in the fourth ventricle. The foramen of Munro is not infrequently largely dilated, and forms a wide communication between the lateral and third ventricles. The white commissures in the latter are often macerated and distended.

Sometimes only a small part of the ventricles is affected, for example, a horn of the lateral ventricle, the aditus infundibuli, the space between the septum pellucidum, etc. In such cases the fluid is often encapsulated and resembles a cyst. As a matter of course, there may be symptoms of local compression of adjacent structures.

We give the following analyses of the fluid in three cases of chronic hydrocephalus.

	Hilger (1867).	Tidy (1869). A child.	Neuhaus (1874). A child, æt. 13 months.
Water........................	98.775	98.492	99.049
Solid matters..................	1.225	0.518	0.951
Albumin	0.246	0.098	0.181
Fats...		0.010	
Alcoholic extracts		0.063	0.004
Ashes	0.762		
Sodium chloride............ ...	0.397	0.214	0.32
Potassium chloride.............	0.082	0.060	
Potassium sulphate.............	0.032		
Potassium phosphate	0 124		
Sodium phosphate........			0.02
Lime phosphate................			0.01
Other phosphates...............	0.096		
Sodium carbonate		0.002	0.41
Sodium sulphate		0.008	
Specific gravity.................	1006.	1007.6	1007.

Reaction alkaline.

Hilger also found mucin, fibrin, urea, succinic acid, cholestearin, and a leucin-like substance. He also obtained 0.164 of a reducing substance which did not ferment, while Tidy obtained 0.063 sugar. Bock discovered sugar in other œdematous fluids. Hoppe-Seyler found sugar which fermented, and deflected polarized light to the right, only after the hydrocephalus had been punctured several times.

Huguenin assumes that more than twenty-five per cent albumin in the fluid of acute hydrocephalus indicates its inflammatory origin.

III. Symptoms.—The most striking external phenomena are observed in children, especially in chronic hydrocephalus. Acute hydrocephalus runs its course so rapidly that sufficient time does not elapse for marked distention of the skull (macrocephaly). Characteristic symptoms are not infrequently absent in acute hydrocephalus, on account of the prominence of the symptoms of the primary disease, especially if the latter gives rise to increase of intracranial pressure.

In chronic hydrocephalus the increase in the size of the skull produces great deformity. In the upright position the head often totters to and fro, or from one side to the other, so that the patients must support it with the hands.

The mental faculties are often affected. The patients become demented, do not learn to speak, or speech is indistinguishable, they pass urine and fæces in the clothes, and must be fed by others.

In addition, we notice epileptiform attacks, contractures, twitchings in individual muscles, pareses, more rarely paralyses, and often spastic symptoms.

If enlargement of the skull is wanting, the symptoms may simulate those of tumor of the brain, and even choked disk may be present.

In some cases optic nerve atrophy and blindness are present from the beginning.

The disease may kill in a few months, but in other cases it continues from childhood until the age of fifty years. Death may occur after an apoplectiform or epileptiform attack, or after coma. Spontaneous discharge of the fluid is occasionally observed, generally through the nose, but also through the mouth, ear, or orbit. Leber described a case in which for five years the fluid trickled constantly from the nose, the amount varying from four to twenty-two centimetres in an hour. This is sometimes followed by spontaneous recovery; in other cases the fluid again accumulates, general and local symptoms of increased cerebral pressure reappear, and finally death occurs.

IV. Diagnosis.—The diagnosis is easy if the size of the skull is increased; if not, it is hardly possible to distinguish the condition from tumor of the brain.

V. The Prognosis is unfavorable, although slight effusions may undergo absorption.

VI. Treatment.—Our attention should first be directed to the primary disease, and then symptomatic treatment is indicated. Very little can be expected from the use of potassium iodide, diuretics, drastics, or blisters. Surgical measures will be considered in the discussion of congenital hydrocephalus.

b. Congenital Hydrocephalus.

I. Anatomical Changes.—This condition almost always occurs as internal hydrocephalus. The amount of fluid may be very large, and it is said that even more than twenty pounds have been found. It is almost always clear and serous in character.

In the fluid withdrawn by puncture, Schlossberger found:

Water,	99.07
Solid constituents,	0.93
Albumin,	0.30
Extractive matters,	0.03
Salts,	0.60

The following was the result of the analysis in Bostock's case:

Water,	98.26
Solid constituents,	1.74
Albumin,	0.6
Sodium chloride,	0.7
Urea,	0.3
Soda,	0.14

As a matter of course, large accumulations of fluid must give rise to serious changes in the skull and brain. The ventricles are enormously dilated, the basal ganglia flattened, the commissures distended, or even torn, etc. The brain often forms a flabby vesicle, whose walls are only a few millimetres in thickness, and in which the gray and white matter are hardly distinguishable. The skull often attains remarkable dimensions, the fontanelles and sutures are widely separated, although later they may undergo ossification.

Congenital hydrocephalus is often associated with other malformations, such as meningocele, encephalocele, hydroencephalocele, hare-lip, club-foot, etc.

II. ETIOLOGY.—In the majority of cases no cause is discoverable. Among those mentioned are drunkenness and syphilis of the parents, injury during pregnancy, and congenital rachitis. Several children, suffering from congenital hydrocephalus, are sometimes born in succession, so that heredity seems to play a part. Some authors attribute it to ulceration of the os uteri during pregnancy, which is said to interfere with the circulation of blood in the fœtal skull. In very rare cases, it is the result of congenital intracranial tumors, with pressure on the blood-vessels.

III. SYMPTOMS.—In some cases the children are born with an hydrocephalic skull, and this may constitute a great obstacle to delivery. The infants are sometimes born prematurely.

In other cases, the external signs of hydrocephalus are very slight, or entirely absent at birth, but after the lapse of days or weeks the growth of the skull is almost visible from day to day. The symptoms are identical with those of the acquired form. Amaurosis from congenital atrophy of the optic nerve has been observed in a few cases.

IV. The DIAGNOSIS is usually easy; the PROGNOSIS is unfavorable, although life is sometimes prolonged to the age of fifty years. Treatment includes the use of diuretics, drastics, derivatives, compression of the head by means of strips of adhesive plaster, and puncture. As a general thing, we must remain satisfied with general treatment.

11. *Hypertrophy of the Brain.*

I. ETIOLOGY.—This condition is most frequent during childhood, and then is generally congenital. It is either well advanced at birth, or it develops gradually after birth. Cases belonging to the latter variety are apt to be regarded as acquired.

Similar changes are sometimes observed in the parents and children, so that heredity seems to play a part. Rachitis has also been regarded as an etiological factor.

In adults, alcoholism, mental strain and excitement, epilepsy and idiocy, even injury, are regarded as occasional causes of this condition.

II. ANATOMICAL CHANGES.—The changes affect chiefly or exclusively the cerebrum, more rarely the cerebellum. They are almost always bilateral, though sometimes only one hemisphere is affected, or only the pons, medulla, or certain

of the basal ganglia. It affects the white matter alone, and is the result of increase of the neuroglia.

When the calvarium and dura mater are removed, the brain presses outwards as if it had not had sufficient room within the skull. The meninges are unusually thin and anæmic, and there is no cerebro-spinal fluid. The surface of the brain is flattened, the brain-substance is anæmic, and the boundary between the white and gray matter is often indistinct. The consistence of the brain is increased and is like that of coagulated albumen, cheese, or tough gum. In some cases, the brain could be stretched like gum without tearing. Upon transverse section of the brain, we are struck with the enormous development of the white matter, the narrowness of the ventricles, and the absence of ventricular fluid.

If the hypertrophy develops at or immediately after birth, the skull also increases in size, and may even attain the dimensions of the hydrocephalic skull. In children in whom the sutures have united, the pressure of the hypertrophic brain may again cause separation of the cranial bones. Some of the cranial bones are occasionally perforated, particularly the roof of the orbit and the sphenoid. In adults, the skull is not distended, but the bones are thinned.

Hyperplasia of the lymphatic glands, thyroid, and thymus glands has been described in several cases of congenital hypertrophy of the brain.

III. SYMPTOMS.—The condition sometimes remains latent. Certain cases were marked by premature and very marked mental development; in others, the chief symptom was enlargement of the skull, which gave rise in children to uncertain gait. Epileptiform convulsions are frequent, and many patients complain of headache, dizziness, and vomiting. Paralyses, contractures, more rarely sensory disturbances, are also observed. Irregularity of the respiration and retardation of the pulse may develop as the intracranial pressure increases. Disturbances of sight have also been mentioned.

The mental faculties are often affected, and apathy, finally dementia, gradually develop.

The symptoms sometimes develop suddenly, and quickly terminate in death, so that the condition has been called acute hypertrophy of the brain, although it had evidently lasted a long time, and the acute symptoms were the result of rapid increase of intracranial pressure. The disease generally lasts many years. Death occurs after convulsions, meningitic symptoms, coma, or apoplectiform attacks.

IV. DIAGNOSIS.—The diagnosis is hardly possible if the skull is not enlarged. In the latter event, it must be distinguished from hydrocephalus. If the skull is translucent, we have to deal with hydrocephalus. In the latter condition, furthermore, the greater fontanelle is convex externally and pulsates less distinctly than in hypertrophy of the brain, in which the fontanelle is flat.

V. The PROGNOSIS is unfavorable, the TREATMENT purely symptomatic.

12. *Atrophy of the Brain.*

I. ETIOLOGY.—Atrophic changes may be congenital or acquired; they affect the entire brain, one-half, or small parts of the organ.

Congenital atrophy of the brain is sometimes the result of imperfect development, sometimes it is owing to involution of parts already developed. Among the causes mentioned are fœtal inflammation of the meninges and ependyma of the ventricles, abnormal distribution of the vessels, premature ossification of the sutures, traumata during pregnancy, and uterine diseases with secondary circulatory changes in the fœtus.

A relatively frequent form is *unilateral atrophy of the brain in children*, which may be congenital or develops soon after birth. One cerebral hemisphere, generally the left, may alone be affected, or the cerebellum is also involved (not infrequently upon the side opposite to the cerebral atrophy), or individual lobes of the cerebrum are chiefly attacked.

General atrophy of the brain is not infrequent in old age (*senile atrophy of the brain*). It must be remembered, however, that this is not a necessary sequence of old age. Closely allied to this variety is the marantic form which develops after protracted diseases (phthisis, cancer, syphilis, Bright's disease).

The atrophy is sometimes the result of diseases of the brain, meninges, or bones of the skull (cerebral hemorrhage, embolism, thrombosis, abscess, tumors, hydrocephalus, meningitis). This variety of atrophy may be confined to the immediate vicinity of the morbid process, or it may extend over a greater area.

Peripheral diseases sometimes result in secondary atrophy of their central terminal stations in the brain. For example, atrophy of the corpora quadrigemina has been observed in phthisis bulbi, and secondary atrophy of the brain has also been known to follow amputations.

Toxic cerebral atrophy is a special form which has been observed as the result of abuse of alcohol, working in lead, and the opium habit.

It is also said that ligature of one carotid may produce unilateral atrophy of the brain, particularly if there are abnormalities in the communications of the circle of Willis.

II. ANATOMICAL CHANGES.—In general atrophy, the brain does not fill the skull-cap; the cerebro-spinal fluid in the subarachnoid space and ventricles is considerably increased. The convolutions are unusually narrow, the sulci deep and broad. The consistence of the brain is generally increased, the cortex has a deep grayish-red color, the white substance is yellowish.

Within the brain, wide spaces are often found around the vessels (état criblée), particularly in the basal ganglia; occasionally we find new-formed cystic spaces filled with serum. The ventricles are very narrow (sometimes the reverse), and the basal ganglia are unusually small. The ependyma of the ventricles is occasionally thickened and covered with warty granulations.

In unilateral atrophy of the brain in children, the hemisphere is sometimes converted into a thin covering, which contains more fibrous than nervous constituents. There may be merely indications of the convolutions. There is often asymmetry of the skull and diminution in size upon the atrophied side, with thickening of the bones. The atrophy is sometimes followed by secondary degeneration, which extends into the spinal cord and even into the nerve roots.

III. SYMPTOMS AND DIAGNOSIS.—In unilateral atrophy of the brain of childhood, the mental and bodily powers rarely remain intact. As a rule, the children are imbecile or idiotic. There are often paralysis and atrophy of the limbs and even the face upon the side opposite to the atrophy; contractures are very often present. The paralysis is less marked in the leg than in the arm. The contractures generally increase on attempting to execute a movement. The atrophy affects chiefly the muscles and bones, and the limbs retain the dimensions of those of a child even after the patient has arrived at adult life. The skin sometimes presents an unusual development of the panniculus adiposus. Sensibility is often very little affected, but there are sometimes disturbances of special sense. Epileptiform convulsions occur frequently. The patients have slight powers of resistance, and readily succumb to intercurrent diseases, or die in an epileptiform, apoplectiform, or comatose attack.

Senile cerebral atrophy is regarded as the cause of the feebleness of memory, childishness, tremor of the limbs, etc., which are peculiar to old age.

Some authors attribute the symptomatology of lead encephalopathy to saturnine atrophy. The tremor, weak memory, and paresis of drunkards are attributed to alcoholic atrophy of the brain.

IV. PROGNOSIS AND TREATMENT.—The prognosis is unfavorable, the treatment purely symptomatic. In unilateral cerebral atrophy of childhood, good results are said to have been obtained from faradism of the paralyzed muscles and gymnastic exercises.

B. DISEASES OF THE MENINGES.

1. *Internal Hemorrhagic Pachymeningitis. Inflammatory Hemorrhagic Changes on the Inner Surface of the Dura Mater.*

ANATOMICAL CHANGES.—This is not a rare condition. In the initial stage, it appears as more or less recent extravasations upon the inner surface of the dura mater, which can readily be removed. In more advanced cases, it forms thin, web-like deposits, which are infiltrated with extravasations and are organically united to the dura mater, sometimes by means of vessels. Older extravasations have a rusty brown color, or impart a diffuse yellow color to the new membrane.

In many cases the process exhibits a progressive character. This is owing to the numerous blood-vessels contained in the new membrane,

and which give rise to fresh extravasations upon the inner surface which is directed towards the cortex of the brain. These extravasations are converted gradually into new membranes. In this manner layer is deposited upon layer, sometimes to the number of twenty or more.

In not a few cases, large extravasations take place between the individual lamellæ (hæmatoma duræ matris). They may attain the size of a fist or more, and compress the brain over a wide extent. The brain sometimes contains a deep depression in which the hæmatoma is situated, or if the hæmatoma is unilateral, the falx cerebri is pushed into the other half of the cranial cavity. The amount of extravasated blood sometimes exceeds sixteen ounces.

On section through the sac of blood, it is often evident that between the different lamellæ are collections of blood of varying age, some fluid and bright red, others coagulated, brownish-red, or rust colored. The walls of these cystic spaces are sometimes smooth and look like a serous membrane, sometimes they are jagged. In some cases their contents are serous, rarely purulent or calcareous.

The new membranes may be adherent to the arachnoid. The latter and the pia mater are occasionally opaque and thickened. The compressed portions of the brain present yellow softening, and the brain is almost always anæmic. The blood sometimes breaks through the lamellæ and destroys the brain tissue.

The changes in question are generally bilateral, and are apt to be located at the top of the parietal region near the falx cerebri, then over the forehead, less frequently near the occiput. They are much rarer at the base of the skull. They are sometimes so extensive as to surround half, sometimes even the entire surface of the brain.

The older physicians believed that the hemorrhages upon the inner surface of the dura mater were primary, and that later they become organized and converted into new membranes. Then Virchow attempted to show that the primary process was an inflammation upon the inner surface of the dura mater, that this results in the formation of a vascular tissue, the rupture of whose vessels produces the hemorrhages.

Recently there has been a return to the older doctrine. Sperling showed, in experiments on rabbits, that the injection of blood under the dura mater terminates, at the end of two to three weeks, in the formation of new membranes, which are exactly similar to those of internal hemorrhagic pachymeningitis in man. Irritation of the dura mater resulted in inflammation, sometimes in suppuration, but never in hemorrhage.

According to Huguenin, the primary hemorrhage takes place from the veins which pass from the convexity of the brain to the longitudinal sinus. Perhaps it also occurs from the veins in the Pacchionian bodies. In some cases, degeneration of the vessels, rupture, and thrombosis have been found. It must be admitted that the theory of the organization of primary hemorrhages in hemorrhagic pachymeningitis is very plausible, since it often occurs under conditions which favor the production of small hemorrhages in other localities.

So long as free coagula alone are found on the inner surface of the dura, they contain nothing but red and white blood-globules, imbedded in a fine network of fibrin. In places the white blood-globules are collected into clumps. The epithelium of the adjacent dura and arachnoid is unchanged. The red blood-globules gradually degenerate and disappear, while the white globules are converted into connective-tissue corpuscles. The tissue in which they lie becomes changed so that it grows cloudy on the addition of acetic acid. The coagulum then assumes gradually the characters of a membrane. In many places the white blood-globules are arranged in rows and result in the formation of capillaries. The latter have an unusually large lumen, possess numerous ramifications and communications with one another, and have unusually thin walls. They often present ampulla-like or rosary-like dilatations. These vessels are supplied from the dura, in places from the arachnoid, from which vessels enter the new-formed mem-

brane. The tendency to hemorrhages arises very early. According to Rind-fleisch, this is explained by the retraction of the newly formed connective tissue, which occludes a portion of the capillaries, and thus increases the blood pressure in the remaining open ones. Hemorrhages are recognizable for a long time as patches of pigment (hæmatoidin), which appears as granules, rhombic tablets and needles, or as a diffuse imbibition. Perls found that they furnished an iron reaction. When the blood-vessels and dura or arachnoid penetrate into the new membranes, there is an intimate connection between the structures, but even then the endothelium of the meninges is intact over large surfaces, so that a diffuse organic union is absent for a long time.

II. ETIOLOGY.—The disease is more frequent in men (in 77.4% according to Durand-Fardel). It is chiefly a disease of advanced life, particularly beyond the age of fifty years. Cases rarely occur in childhood (they have been observed at the age of four, six, seven, and nine months). Among the direct causes may be mentioned:

  a. Traumata; the period between the injury and the first manifest symptoms may last several (three) years.

  b. Inflammations conveyed from the vicinity, for example, in tuberculosis of the petrous portion of the temporal bone, and bones of the skull in general.

  c. Abuse of alcohol.

  d. Diseases of the brain: tumors, softening, particularly atrophy; insanity, almost constantly in dementia paralytica.

  e. Infectious diseases: pleuropneumonia, typhoid and typhus fever, relapsing fever, acute articular rheumatism, small-pox, scarlatina, whooping-cough, pyæmia, puerperal fever.

  f. Diseases of the respiratory and circulatory organs: phthisis, pleurisy, pneumothorax, pericarditis, valvular lesions, atrophy of the heart muscle.

  g. Marasmus and conditions of blood dissolution: cancer, Bright's disease, progressive pernicious anæmia, leukæmia, hæmophilia, and scurvy.

  h. Congenital and acquired syphilis has been mentioned among the causes.

  In not a few cases no cause can be discovered.

  Until recently it was generally believed that pachymeningitis was the result of conditions of arterial fluxion, especially in the middle meningeal artery. But Huguenin attaches chief weight to changes in the blood-vessels which predispose to abnormal brittleness or permeability. In many cases, in addition, there is stasis (cough, heart diseases), but in the majority there is diminution in the volume of the brain which favors the occurrence of hemorrhages.

  III. SYMPTOMS AND DIAGNOSIS.—Morbid symptoms are absent in many cases of internal hemorrhagic pachymeningitis. The extravasated blood and new membranes take up so little room that they produce no noteworthy compression. It is very probable that, in many cases, they give rise to headache, but a diagnosis cannot be based on such a vague symptom alone. Fuerstner has recently called attention to constantly recurring, apparently purposive grasping movements, which are often observed in somnolent individuals, for example, in phthisical individuals during the agony, and in whom the autopsy discloses internal pachymeningitis. The patients are constantly pulling the beard, genitals, bed covering, etc. Fuerstner regards these symptoms as the result of irritation of the motor cortical centres by the hemorrhage.

  More marked symptoms generally appear as evidences of sudden in-

crease of pressure in the cranial cavity, and generally affect the convexity of the brain to the most marked degree. Such symptoms occur only after larger extravasations of blood.

As a rule, an apoplectic seizure develops. The patients fall unconscious, remain comatose for hours, days, and even weeks, or death occurs in coma at the end of a longer or shorter period. The coma is followed not infrequently by a dream-like condition. The patients sleep a good deal, pass urine and fæces in bed, must be aroused to eat and drink, do not know where they are, and stagger in walking. The pupils are narrow. Later, they become moderately dilated, but are rigid, and not very sensitive to light. The pupils are sometimes unequal, the wider one being generally on the side opposite to the lesion. In three cases Fuerstner observed choked disk, as the result of entrance of blood into the optic sheath; he also observed unilateral nystagmus. Paralysis of ocular muscles is almost always absent. Paralyses are frequent symptoms. All the limbs are often paretic, one side more markedly than the other, and the facial and hypoglossus nerves may also be implicated. Occasionally there are irritative symptoms, such as convulsions and contractures of certain limbs. Forced movements of the eyes, head, and body are sometimes noticed. Among the most frequent symptoms are slowness and irregularity of the pulse. Fuerstner mentions profuse diaphoresis. Considerable rise of temperature has also been described (41° C.).

The symptoms may gradually subside, if the extravasated blood is partly absorbed, and the brain accommodates itself to the increased pressure.

Per se the symptoms described are not indicative of hemorrhagic pachymeningitis, since they are merely evidences of sudden increase of intracranial pressure. But the matter is different if the etiology favors the view of the development of an hæmatoma, and if such attacks recur from time to time. In all cases, however, the diagnosis is difficult, and often very doubtful.

In psychopathic conditions, hæmatomata may develop without giving rise to the symptoms mentioned above. They are often noticeable only from the peculiar change in the psychical condition, beginning with excitement and then passing into coma.

IV. PROGNOSIS AND TREATMENT.—The prognosis is always serious, and so much the more the more pronounced the symptoms of compression. Nor are we able to prevent the occurrence or return of the hemorrhages.

During the apoplectic attacks, venesection sometimes relieves the coma with great rapidity, but it should not be performed in feeble individuals. An ice-bag should be applied to the head, and laxatives administered, for example: ℞ Inf. sennæ comp., ʒ vi.; sodii sulph., ʒ vi. M. D. S. One tablespoonful three or four times a day. Later we may order absorbents, particularly potassium iodide (gr. xij. t. i. d.). The galvanic current may also be employed. During the acute period, some physicians order leeches, blisters, irritating ointments to the forehead, mastoid processes, or nape of the neck.

2. *Thrombosis and Inflammation of the Cerebral Sinuses.*

I. ETIOLOGY.—These conditions are found not infrequently. But however close their relations to one another, they must be strictly sep-

arated anatomically and clinically.   Inflammation of the cerebral sinuses is almost always followed by secondary thrombosis, and hence the clinical symptoms will be identical in so far as they depend on disturbances of venous circulation.

Pure (non-inflammatory) thrombosis is generally of marantic origin. The cerebral sinuses are particularly adapted to the formation of thrombi, because their walls are fixed, and adapt themselves with difficulty to variations in the amount of blood; their lumen is often angular, and they are often traversed by threads and excrescences (Hyrtl).   Marantic thrombi are found most frequently in the superior longitudinal and transverse sinuses.   They occur often in children who are enfeebled by chronic diarrhœa, suppuration, chronic pulmonary diseases, etc.   Under similar conditions the thrombi may also form in adults.

Compression thrombi are observed much more rarely.   The causes of compression may be situated in the cranial cavity (brain and meningeal tumors), or they are extracranial.   In the latter event, they affect mainly the internal jugular vein, more rarely the superior vena cava (lymphatic glands or mediastinal tumors).   The thrombi in the latter vessels then spread upwards to the transverse sinus, more rarely to the inferior petrosal sinus.

Whether thrombosis of the sinuses may develop as the result of mere stasis of blood and slow circulation is not known with certainty.

Inflammation of the cerebral sinuses with secondary (inflammatory) thrombosis is most frequently the result of inflammation near the dura mater.   It is observed most commonly in tuberculous processes in the petrous portion of the temporal bone, which gradually extend through the bone to the dura mater.   Purulent inflammations of the meninges and brain substance also extend occasionally to the cerebral sinuses.   In some cases the primary site of inflammation is far removed, and the sinuses are affected, either as the result of thrombo-phlebitic processes which extend from extracranial veins into the sinuses, or the blood carries infectious and inflammation-producing particles into the sinuses.   To this category belong wounds of the skull and face, which may be very trifling at the start, until suddenly the symptoms of thrombosis of the sinuses give evidence of great danger.   Cases have been observed after abscess of the gums, facial erysipelas, diphtheria, abscesses in the deep muscles of the neck, orbital inflammations.

In very rare cases, it is the result of direct traumatic inflammation. Among one hundred and fifty-one cases of thrombosis and inflammation of the sinuses collected by Wreden, one was the result of the entrance of a foreign body through the spheno-orbital fissure.

II. ANATOMICAL CHANGES.—In simple thrombosis, the sinus contains a brownish-red, rusty brown, or grayish-red clot which is notably firm.   It is generally adherent to the wall of the sinus, to which it is sometimes organically united.   On transverse section, it not infrequently presents distinct lamination.   Upon the primary thrombus are sometimes deposited others, whose increasing age is shown by the greater discoloration and consistence.   The primary thrombi sometimes occlude the vessel entirely, or they are situated only upon its walls.   They may fill the entire sinus and even extend into the adjacent sinuses and veins.   Thus in thrombosis of the superior longitudinal sinus the clots sometimes extend into the veins which pass into the sinus from the convexity of the brain.   Thrombosis of the transverse or inferior petrosal sinus often extends to the internal jugular vein and gives rise, during life, to collapse

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of the external jugular on the same side (Gerhardt). In other cases, the thrombosis affects only a small part of the sinus, or the latter contains several thrombi.

The changes described are associated with venous stasis on the peripheral side of all the vascular tracts which supply blood to the affected sinus. These symptoms of stasis will be most marked in bilateral complete occlusion of the transverse and inferior petrosal sinuses, with continuation of the thrombus into the internal jugulars. They are manifested by marked venous congestion and hemorrhages into the brain and meninges, followed by cerebral softening, by increase and sometimes bloody discoloration of the cerebro-spinal fluid, and by hydrocephalus ventriculorum.

Particles of a thrombus are sometimes dislodged and enter the right auricle, ventricle, and pulmonary artery, and are thus conveyed to the lungs, where they constitute pulmonary emboli (hemorrhagic, wedge-shaped infarctions).

Some authors maintain that thrombi may undergo organization and be partly absorbed, or at least become permeable and permit the passage of blood. This is denied by others.

In inflammation of the cerebral sinuses with secondary thrombosis, the effects upon the venous circulation are the same as in simple thrombosis. In addition, there is a tendency to suppuration and gangrene. The thrombi are not infrequently soft, purulent, ichorous, and foul smelling. The wall of the sinus is infiltrated, brittle, sometimes on the point of rupture. There may be inflammation of the dura mater and other meninges, also abscess of the brain. Pulmonary emboli give rise to secondary inflammation and metastatic abscesses, and these may rupture into the pleural cavity. The primary affection may also have given rise to pyæmic foci of pus in the spleen, liver, and kidneys.

III. Symptoms and Diagnosis.—The recognition of thrombosis of the sinuses is very difficult, and often impossible.

In not a few cases no symptoms are observed. This is especially true if previous disease has enfeebled the circulation and diminished the irritability of the brain to such an extent that the increment of circulatory disturbances produces no noteworthy effects.

Bouchut maintains that many cases of convulsions in children, occurring just before death, depend upon thrombosis of the sinuses, but this symptom is too vague to warrant even a probable diagnosis.

A third series of cases is attended with clouded consciousness, inequality of the pupils, strabismus, nystagmus, rigidity of the back of the neck, vomiting, convulsions, paralyses, and contractures. But these symptoms and the primary disease are more apt to arouse the suspicion of purulent meningitis, and, indeed, this lesion is generally present while the thrombosis remains unrecognized.

Disturbances of central innervation are sometimes absent and all else is masked by the symptomatology of the pyæmic condition.

A probable diagnosis may be made if the symptoms of pulmonary infarctions or metastatic abscesses make their appearance, while, at the same time, diseases are present which, as a matter of experience, are known to give rise to thrombosis of the sinuses, and other causes of pulmonary embolism are absent.

The diagnosis grows more positive if there are evidences of stasis in the external veins of the face, scalp, and neck.

In thrombosis of the superior longitudinal sinus, there is unusual dis-

tention and sinuosity of the veins which are situated between the greater fontanelle and the temporo-auricular region. This is especially distinct in children in whom the emmissaria Santorini are very large, and the hair on the scalp is scanty. In a girl, æt. 3 years, under my observation, the distention of the veins was so marked as to give rise to gross deformity. According to Gerhardt, there is also cyanosis in the distribution of the anterior facial veins. v. Dusch observed epistaxis as the result of stasis in the communications between the veins of the nose and the superior longitudinal sinus, but Staeger observed the same symptoms in thrombosis of the cavernous sinus. An important feature in children is the condition of the fontanelle. The greater fontanelle is depressed at first so that one parietal bone is sometimes pushed over the other. Later, the fontanelle becomes more extensive and tense than it was originally, if the cerebro-spinal fluid increases as the result of occlusion of the sinus.

In thrombosis of a transverse sinus, the corresponding external jugular vein is sometimes emptier than the one on the healthy side, because it empties its blood more readily into the less distended internal jugular. This symptom will be so much more marked the more vigorous the circulation, and the further the thrombus extends into the beginning of the jugular vein (which receives the inferior petrosal sinus), or if the superior and inferior petrosal sinuses are both occluded.

If a thrombus of the transverse sinus extends to the posterior auricular veins, œdema (hard and tender) develops behind the ear and mastoid process.

If both transverse sinuses are occluded, the symptoms of thrombosis of the superior longitudinal sinus may be produced.

In thrombosis of the cavernous sinus, the chief symptom is stasis in the orbit, since this sinus receives the blood of the ophthalmic vein (œdema of the lids and conjunctiva, acute exophthalmus from distention of the retrobulbar veins, occasionally œdema of an entire half of the face (stasis in the communications between the ophthalmic and facial veins), venous congestion of the retina (sinuosity and distention of the veins and narrowing of the arteries), œdema of the retina, choked disk, amblyopia or amaurosis). Bouchut described thrombosis of the retinal veins.

Another important symptom of thrombosis of the cavernous sinus consists of disturbances of innervation of the trigeminus (first branch), trochlear, oculomotor, and abducens nerves, which pass between the sinuses, and are apt to be irritated or paralyzed by stasis-œdema of the surrounding connective tissue (trigeminal neuralgia, ocular paralysis, trophic changes in the eye).

Similar symptoms may be looked for, if both inferior and transverse petrosal sinuses are obstructed.

Thrombosis of the sinuses generally runs an acute course ; death often occurs in a few days. The average duration is one to four weeks. Recovery is of rare occurrence. There are sometimes striking remissions and exacerbations, but the former should not induce us to make a premature favorable prognosis. Elevation of temperature occurs in simple thrombosis, remittent and intermittent fever in purulent thrombosis. Death is the result of increasing exhaustion or excessive depression of the central nervous system.

IV. PROGNOSIS AND TREATMENT.—The prognosis is almost always unfavorable. The treatment is purely symptomatic.

3. *Meningeal Hemorrhages.*

I. ANATOMICAL CHANGES.—Hemorrhages may be situated between the meninges or in the tissues of the individual meninges.

According to their situation we ·distinguish epidural, subdural, subarachnoidal, and subpial hemorrhages; several forms are often combined.

Fig. 121 furnishes a schematic representation of the topographical relations of the meninges. The dura mater is intimately adherent to the inner surface of the bones of the skull (endocranium). Hemorrhages between the skull and the adjacent outer surface of the dura mater are called epidural hemorrhages. They can only occur if the blood separates the dura from the bones (vide Fig. 121, 1. *epr*).

Between the dura mater and arachnoid is found a capillary space (subdural space, 2. *sdr*), corresponding to the arachnoid cavity of older writers. Hemorrhages into this cavity are called subdural hemorrhages.

Between the arachnoid and pia mater is a meshed tissue (Fig. 121, 3. *sar*) in which the cerebro-spinal fluid is situated. It also contains the large cerebral arteries and veins. Hemorrhages in this locality are called subarachnoid hemor-

FIG. 121.

Longitudinal section through the bones, meninges, and surface of the brain (schematic). 1. *epr*, epidural space ; 2. *sdr*, subdural space (arachnoid cavity of older writers ; 3. *sar*, subarachnoid space ; *sk*, bones of the skull ; *dm*, dura mater ; *ad*, arachnoid with subarachnoid tissue ; *pm*, pia mater.

rhages. Finally, intracerebral hemorrhages which extend to the surface beneath the pia mater constitute subpial hemorrhages. The pia is often ruptured, and the blood then passes into the subarachnoid space.

Meningeal hemorrhages vary in extent and number. They may be multiple, many of them attaining barely the size of a pin's head, or there may be a single large extravasation. The latter is sometimes so considerable as to surround the entire brain, fill the ventricles, and extend along the spinal canal to the filum terminale; in other cases, it is confined chiefly to one-half of the brain, or to the convexity or base. The appearance of the blood varies according to the age of the hemorrhage. If it is followed very rapidly by death, the blood is fluid or forms loose, dark-red clots; in other cases it is brownish-red, rust colored, and firmer in consistence. When the hemorrhage occurs in repeated relapses, a sort of lamination is produced, the older brownish masses being situated externally, the more recent, red ones internally. Hemorrhages of slight extent seem to be capable of undergoing absorption, and to this are attributed the pigmentations, thickenings, and adhesions of the meninges to each other and to the surface of the brain (sequelæ of inflammatory reaction).

As a matter of course, hemorrhages of a certain extent must compress the cranial contents. The surface of the brain beneath the extra-

vasation is flattened, and the cortex, on section, is pale, and its consistence is increased. If the hemorrhage has lasted for some time, the surface of the brain is generally œdematous. There is often destruction of brain tissue, as the result of the primary injury.

II. ETIOLOGY.—The causes of meningeal hemorrhages are traumatism, circulatory disturbances, and changes in the constitution of the blood.

Traumata include a blow, fall or cut upon the head, or severe concussion of the entire body. The hemorrhages may occur on the side of the injury, or on the opposite side, and may or may not be associated with injury to the soft parts and bones.

Traumatic meningeal hemorrhages are not infrequent in the new-born, as the result of too rapid or too protracted labor, application of the forceps or narrow pelvis. These hemorrhages are the result of marked displacement of the meninges upon the bones of the skull.

Circulatory disturbances act as a source of meningeal hemorrhages, for example, in thrombosis of the sinuses, rupture of aneurisms of the cerebral arteries at the base of the brain (most frequently the basilar or middle cerebral artery). In rare cases meningeal hemorrhages occur as the result of stasis in cardiac and pulmonary diseases, often in spasmodic conditions (tetanus, trismus, epilepsy), but in the latter event, it is often doubtful whether the hemorrhage is primary or secondary.

Meningeal hemorrhages as the result of changed constitution of the blood are found not infrequently in infectious diseases, leukæmia, scurvy, hæmophilia, progressive pernicious anæmia, grave jaundice, etc.

III. SYMPTOMS AND DIAGNOSIS.—Slight meningeal hemorrhages may present no symptoms and may be discovered accidentally on autopsy. Large hemorrhages in the new-born often run a latent course. Gerhardt believes that this is owing to the imperfect development of the motor cortical centres.

In some cases, particularly in children, the hemorrhage produces epileptiform and tetanic spasms; but, as a rule, the diagnosis cannot be made.

In the new-born, meningeal hemorrhages are sometimes the cause of asphyxia neonatorum. The little ones breathe very poorly or not at all after delivery, have a cyanotic or leaden-gray appearance, are somnolent, and die in a few minutes, hours, or days. Their condition occasionally is tolerably good immediately after birth, but in a few days the above-mentioned symptoms make their appearance, and the patients perish. The symptoms are explained in part by renewed and more extensive hemorrhages.

Meningeal hemorrhage often co-exists with intracerebral hemorrhage, but we are unable to determine during life whether the latter exists alone or in combination with the former.

The symptoms occur in their purest form when the hemorrhage is the result of rupture of an aneurism. They consist chiefly of the evidences of acute increase of intracranial pressure, such as we described in the discussion of internal hemorrhagic pachymeningitis. The patients fall unconscious, either suddenly or after various prodromata (dizziness, headache, vomiting), the respirations are irregular, the pulse slow, the pupils narrow, and usually react slowly or not at all, urine and fæces are passed involuntarily; there is generally paralysis or rigid contracture of all the limbs. Death occurs very rapidly (within the first ten hours in seventeen out of forty-eight cases collected by Lebert), or the patients recover consciousness, are paralyzed, and die in a few days, after,

increasing symptoms of cerebral paralysis. Well-defined hemiplegia is rare, even if the hemorrhage is unilateral.

Recovery can be looked for only after small hemorrhages.

IV. TREATMENT.—The treatment is purely symptomatic: ice-bag to the head, stimulants; in asphyctic new-born infants, stimulation of respiration by cutaneous irritants and faradization of the phrenic nerve, administration of milk and wine; later, absorbents.

C. FUNCTIONAL DISEASES OF THE BRAIN.

(CEREBRAL NEUROSES.)

1. *Epilepsy.*

I. ETIOLOGY.—Epilepsy is a chronic disease, the typical form of which appears as attacks of unconsciousness and clonic convulsions.

The disease is very frequent; according to different authorities, there are one to six epileptics to one thousand individuals. It was formerly supposed to be more frequent among males, but this has been denied. The disease generally begins between the ages of seven to twenty years. But cases of congenital epilepsy have been reported, and in rare instances it begins at a very advanced age (seventy years).

Epilepsy may be primary or idiopathic, and secondary or symptomatic. The former is an independent affection and chronic neurosis of the brain, the latter is excited by changes in various parts of the body.

Heredity plays a prominent part among the causes of primary epilepsy.

In some families, it is hereditary; in others, hysteria, insanity, neuralgia, etc., alternate in different generations. In some cases, certain generations escape the neuropathic taint. It has been maintained that epilepsy is more apt to be inherited from the mother. Phthisis has also been looked upon as the cause of epilepsy in the offspring (?).

In certain cases, it is not so much nervous heredity which comes into play, but other injurious factors in the parents afford the basis for epilepsy in the offspring. This is particularly true of drunkenness.

Epilepsy is sometimes the result of excessive mental excitement (fright, sorrow, excessive joy). This is also true of mental strain.

In rare cases, individuals become epileptic as the result of so-called imitation, *i. e.*, after seeing an epileptic attack in another individual. It is also stated that the disease may develop in persons who have simulated epilepsy for a longer or shorter period.

Bodily strain is also regarded as an occasional cause of the disease.

It sometimes occurs after infectious diseases, particularly syphilis. Indeed, it has been held that this cause should be suspected in all cases of epilepsy which develop after the age of thirty years. It may also follow pleurisy, pneumonia, measles, scarlatina, variola, acute articular rheumatism, and scrofula.

Constitutional changes, such as rickets, are also said to play a part in the etiology.

Of great importance are the development of puberty and various other processes in the sexual life. Many become epileptic at the period of first menstruation, others during the first act of coitus. Some women are first attacked during pregnancy. It is also stated that congenital

phimosis is frequent in epileptics, and there is no doubt that onanism favors the development of the disease. This has also been asserted with regard to sexual continence.

The epilepsy of drunkards must be regarded, to a certain extent, as a toxic form of the disease.

The disease is sometimes the result of injuries which produce diffuse concussion and molecular changes in the central nervous system (fall, blow, etc., upon the skull and spinal column).

Injuries may also give rise to epilepsy in another way, and this leads us to the consideration of reflex epilepsy, and thus of secondary epilepsy.

The term reflex epilepsy is applied to those cases in which the disease is produced by morbid changes situated in regions which are sometimes far distant. For example, cicatrices which inclose nerves, and tumors which compress nerves, may produce such a condition of increased irritability of the central nervous system by their constant irritation as to result in epilepsy. This has also been observed as the effect of an accumulation of wax in the ear, foreign bodies or inflammation of the ear, polypi of the vocal cords and naso-pharyngeal space, hyperplasia of the nasal mucous membrane, intestinal worms, coprostasis, distention of the intestines by indigestible substances, and genital diseases. The epilepsy sometimes disappears after the removal of the reflex exciting cause.

Epilepsy is occasionally the result of lesions of the cranial cavity, such as fracture of the bones and impaction of particles in the brain, tumors, softening, abscesses, and cysticerci in the brain. Many of these cases are the result of direct irritation of the cortical motor centres (cortical epilepsy).

The causes of the morbid predisposition and of the individual attacks must be kept separate. Both often remain unknown; in other cases, the individual attacks are produced by bodily or mental exertion, excesses in Venere et Baccho, suddenly increased irritation, for example, coitus, etc.

II. ANATOMICAL CHANGES.—The anatomical changes which give rise to primary epilepsy are unknown. Some of the assumed anatomical lesions are undoubtedly accidental. The following changes have been found :

Asymmetry of the skull, thickening, calcification, ossification, and pigmentation of the meninges, punctate and larger extravasations in the meninges, brain, and spinal cord, if death occurred during an epileptic attack (the result of stasis in the circulation); stenoses of the occipital and carotid foramina, stenoses and abnormal communications in the circle of Willis, interstitial and parenchymatous changes in various parts of the central nervous system with inflammatory changes in the bloodvessels, atrophy of the cornu Ammonis, hyperæmia, proliferation of interstitial connective tissue and degeneration of the ganglion cells of the sympathetic, etc

III. SYMPTOMS.—We divide epilepsy into two principal forms, severe and mild, according to the intensity and significance of the symptoms.

Severe epilepsy (epilepsia gravis) is recognized most readily when it occurs under the typical history of well-developed epileptic attacks. These may occur suddenly or are preceded by certain prodromata.

The prodromata are either remote or immediate. The former precede the attack for hours or days, the latter often last only a few seconds.

The remote prodromata generally consist of psychical changes. The patients grow moody, irritable, sleepless or somnolent, forgetful and apathetic—symptoms which disappear after the epileptic attack.

The immediate prodromata are also known as aura epileptica, because some patients state they can recognize the onset of a fit by a sensation as if a current of air were blowing upon them. But this form of aura is very rare.

According to the region affected, we distinguish a sensitive, motor, sensory, and vaso-motor aura.

The sensitive aura consists of paræsthesiæ of various kinds: prickling, sensation of cold, drawing pains, feeling of oppression in the præcordia, eructations, distention of the abdomen, etc. The sensations sometimes recur before each attack, sometimes different ones.

The motor aura consists of a feeling of numbness, paralysis, or twitchings in the muscles of the limbs or face. The symptoms sometimes pass from one limb to the others in regular order, until the epileptic fit appears.

The sensory aura manifests itself not infrequently in abnormal special-sense impressions. The patients complain of peculiar sensations of taste or smell, or they hear ringing or roaring in the ears, or see bright flaming colors (often red). Sometimes there are hallucinations, particularly the appearance of frightful shapes, or delirium develops. In rare cases there is a sensation of unspeakable well-being.

The vaso-motor aura appears as pallor, coldness, later as a cyanotic color of the skin, associated with a feeling of falling asleep. The phenomena often begin in one limb and then spread to all the others. The majority of patients state that the phenomena extend from the periphery towards the centre, but they are evidently the result of previously existing disturbances of central innervation.

The aura generally lasts a few seconds or minutes, but its duration may vary considerably in the same individual. In many patients the epileptic attacks sometimes occur with, sometimes without an aura. The latter sometimes occurs without being followed by an epileptic seizure.

The presence or absence of an aura has no noticeable effect upon the number and character of the epileptic seizures. In some cases, the seizure may be prevented by vigorous counter-irritation at the onset of the aura (rapidly strapping the limb from which the aura starts, swallowing a teaspoonful of salt, etc.). But many patients maintain that the artificial prevention of an attack is not infrequently followed for a long time by a feeling of great exhaustion and mental depression, while they feel relieved and refreshed after an attack.

The epileptic attack proper begins, in many patients, with a shrill, heart-rending cry, which is the result of sudden tonic spasm of the muscles of inspiration or expiration. Almost at the same time the patients fall unconscious, and thus often sustain severe injuries. Scars upon the skull or other parts of the body are rarely absent in those who have had numerous attacks. The unconsciousness is complete. Individuals who have fallen into the fire during the attack have been known to be burnt to the bone without experiencing the slightest sensation. At first, the color of the face and skin is pale, and the entire muscular system undergoes tonic spasm. The eyes are turned fixedly upwards and inwards, the features are distorted, the head is drawn strongly backwards, the muscles of the back and limbs are rigidly contracted. The stage of tonic spasm generally does not last more than ten to fifteen seconds. This is followed by the stage of clonic muscular spasms; shortly before the pale color of the skin has generally given place to cyanosis. During the clonic convulsions, rapidly changing contortions and grimaces ap--

pear in the face. The eyes roll to and fro, the tongue is protruded between the teeth and retracted, and the organ may be bitten. The cicatrices left over may be important in diagnosis. The muscles of the pharynx and larynx also take part in the clonic convulsions. The saliva cannot be swallowed, and appears externally as a white, frothy, sometimes sanguinolent mass. Spasm of the muscles of the neck compresses the jugular veins, which appear under the skin as cords almost of the thickness of the little finger. The stasis is often so great that more or less extensive hemorrhages appear beneath the conjunctiva and the integument of the forehead and face. This may also occur in other parts of the body. These subscutaneous hemorrhages are especially important in the diagnosis of epileptic attacks which have occurred during sleep. The stasis of blood also gives rise to protrusion of the eyeballs. The fingers are generally contracted spasmodically, and the thumb drawn forcibly into the palm of the hand. The patient constantly works convulsively with the arms and leg, and may fall out of bed. The respirations are rendered irregular by the spasm of the respiratory muscles, sighing and gurgling sounds are produced, and the cyanosis increases.

The condition of the pupils varies. As a general thing they are markedly dilated and they never react to light. The latter characteristic is important in excluding simulation. Reflex action is abolished, as a rule, but in some cases the lids are closed upon touching the cornea, and muscular twitchings occur upon sprinkling the body with cold water. There is sometimes involuntary evacuation of urine and fæces; priapism and evacuation of semen may also occur.

The bodily temperature is almost always unchanged, at the most it rises a few tenths of a degree. The pulse is sometimes irregular and small; if the convulsions are very violent, it may be imperceptible.

The clonic convulsions are often equally vigorous on both sides of the body, sometimes they are stronger on one side. The vigor of the contractions may be very considerable. They may result in fracture of the teeth, dislocations and fractures of bones. Shortes reports a case of rupture of the heart, probably as the result of the enormous stasis.

The stage of clonic spasm lasts from half a minute to five minutes, rarely longer. Towards the end of the attack the skin is often covered with profuse, usually clammy, cool perspiration, rumbling in the abdomen and eructations often make their appearance, the twitchings become less violent and frequent, and gradually cease. In rare cases the spasms cease abruptly.

This stage is followed by a series of phenomena which are called the post-epileptic stage.

The cyanosis gradually disappears, but the patients remain asleep for a certain period. Many awaken with a deep sigh, unaware of what has happened. For hours, even for days, they remain stupid, forgetful, irritable, and a feeling of well-being and relief appears only gradually. In others delirium sets in. Or there may be maniacal attacks and acts of violence (biting, destructive acts, homicidal attacks) of which the patients have no recollection when they recover complete consciousness. Transitory paralyses are sometimes left over. I recently had under observation an epileptic who suffered, after an attack, from complete right hemiplegia and aphasia. At the end of a week the symptoms rapidly disappeared. In another case the paralysis persisted three weeks. Sometimes there is paresis or paralysis of a single limb.

Huppert claims that transitory albuminuria is a constant post-epileptic symptom, and that hyaline casts and spermatozoa are sometimes found in the urine. This is by no means true of all cases; in twenty cases examined by Ebstein, albuminuria was not found in a single one. Polyuria is sometimes observed.

In some patients, epileptic attacks occur only in the day (diurnal epilepsy), in others only at night (nocturnal epilepsy). Not infrequently they appear at different times in the day. Nocturnal attacks are recognized by the production of extravasations and wounds of the tongue, or the soiling of the bed with urine and fæces, or by the fact that the patients feel remarkably exhausted on awaking. Attacks may occur after intervals of months and even years, of a few days or hours, or even less. In one of Delasiauve's patients 2,500 attacks occurred in a single month. There are not infrequently periods in which the attacks recur at short intervals, then longer or shorter periods in which they remain entirely absent. The attacks often cease during the course of acute infectious diseases, and may even disappear permanently.

The patients are sometimes seized by a new attack before they have entirely recovered from the preceding one. The constant repetition of such seizures for days is known as the *status epilepticus*. As a rule, this is accompanied by great elevation of temperature, which may reach 42° C. or even more, and may prove fatal. Remissions and exacerbations are noticed in some cases, and recovery may occur, but is rarer than the fatal termination. The excessive rise of temperature appears to be the result of central disturbances of innervation.

The sequelæ of the epileptic attack are divided into the immediate and mediate. The former give rise to death or other injury directly as the result of the seizure. Death may occur from suffocation as the result of tonic and clonic spasm of the respiratory muscles, or suffocation results accidentally from the falling of the patient on his mouth and nose so that the air passages are occluded, or from the occurrence of the attack while eating and the entrance of food into the larynx. The serious disturbances of circulation sometimes give rise to pulmonary œdema or cerebral hemorrhage; rupture of the heart is also a possible sequel. The disturbances of less significance include cutaneous wounds and excoriations, fractures and dislocations.

Persons who have long suffered from epileptic attacks often present a change in their bodily and mental characteristics. The features become dull, stupid, occasionally animal, the eyes are prominent and staring, the lips are thickened, etc. Dilatation of the retinal veins as the result of stasis is sometimes observed. The changes in the mental sphere are shown by the irritability, diminished power of memory and judgment. Children are no longer able to make their way in school, dementia or some other form of insanity makes its appearance, and not a few patients end their lives in lunatic asylums. In some cases the mental powers are unaffected, and some of the most celebrated men have suffered from this disease.

Mild epilepsy appears in various forms. In some cases the symptoms are confined to temporary disturbances of consciousness (absentia epileptica). The patients suddenly grow pale, have a staring, glassy look, stop suddenly in the middle of a sentence, or in walking, playing on the piano, or any other occupation in which they may be engaged. At the end of a few minutes the pallor of the face disappears, the patients sigh deeply, or yawn several times in succession, and then continue the oc-

cupation which had been interrupted. In some cases the seizure is followed at once by confusion of ideas and delirium, and some time elapses before the patient comes to himself. It may also happen that they perform unconscious automatic actions during the attack. Such conditions often remain unknown to the patient himself for a long time. Very many seizures sometimes occur during the course of an hour, in others they appear at long intervals.

In some patients there is not complete loss of consciousness, but merely temporary confusion and vertigo (epileptic vertigo). They are generally forced to hold on to some support or to sit down, more rarely they fall to the ground.

In certain cases of mild epilepsy, typical convulsions occur, but they are very brief and slightly developed. Twitchings occur in only a few groups of muscles, sometimes only tonic, sometimes so slightly clonic as to resemble tremor, rather than a clonic muscular spasm. It is also said that the attack may consist merely of muscular spasm without loss of consciousness, but we have never seen attacks of this character.

Some patients suffer only from attacks of mild epilepsy. In others, the disease begins in the mild form, later it merges into the severe variety. The reverse condition may also obtain. Mild and severe attacks may also alternate irregularly with one another.

In addition to typical epilepsy (mild and severe forms), there is also an atypical form of epilepsy, whose epileptoid character is often recognized with difficulty. This includes the morbid states to which Griesinger applied the term epileptoid conditions. The symptoms are often purely psychical. The patients have temporary hallucinations and delirium, talk obscenely, act in a strange manner, or become violent. These symptoms occur in paroxysms, and are not remembered by the patient. They are particularly important from a medico-legal standpoint, since to them homicide and other acts of violence may owe their origin. They often appear suddenly; in other cases, they are preceded by prodromata, so that the patients warn those around them of the approaching danger. In some instances, the symptoms assume more of a motor character; the patients suddenly begin to run forwards or to revolve in a circle, without being conscious of their action. In others, there are sudden outbreaks of perspiration, etc.

IV. PATHOGENESIS.—In a disease which consists of brief, temporary attacks, the causes must be assumed to be transitory, abnormal conditions of the brain, such as temporary circulatory disturbances. These are now generally regarded as anæmic in character.

The epileptic attack undoubtedly starts from the motor regions in the central convolutions of the brain. Hitzig succeeded in making dogs epileptic by extirpation of the motor centres in the cerebral cortex, and the experiments of Unverricht and P. Rosenbach point in the same direction.

It was formerly held that the site of epilepsy is located in the medulla oblongata.

Nothnagel located the so-called convulsive centre in the pons, and explained the occurrence of the epileptic attack in the following manner: irritation of the vaso-motor centre in the medulla oblongata; spasm of the cerebral arteries; anæmia of the brain; loss of consciousness; in addition, irritation of the convulsive centre, general convulsions.

Brown-Séquard found that guinea pigs became epileptic after section of the sciatic, or hemi-section of the spinal cord. Westphal produced epilepsy in guinea pigs by making quick strokes with the hammer upon the head. At the

autopsies, small extravasations of blood were found in the medulla oblongata and upper part of the cervical cord. Both authors noticed that the epileptic attacks did not begin until some time after the experiment, that the individual attacks may be produced by irritation of definite parts of the skin (so-called epileptogenic zone), and that the disease is transmitted to the offspring. Unverricht and Rosenbach have recently produced epileptic attacks in dogs by irritation of the cerebral cortex.

V. DIAGNOSIS.—The diagnosis of the typical forms of epilepsy is generally easy, while a long time may elapse before the true nature of atypical epilepsy is recognized.

Not every epileptiform spasmodic seizure may at once be called epilepsy; it must be remembered that epilepsy is a chronic disease, characterized by the recurrence of the attacks, and that gross, particularly acute, anatomical changes are not discoverable.

In the differential diagnosis between epilepsy and hystero-epilepsy, the chief element is the fact that in the latter consciousness is retained or at least not entirely abolished. In addition, other hysterical symptoms are generally present.

Epilepsy is not infrequently simulated. But in malingerers no wounds or cicatrices are found upon the tongue or skin, the pupils react to light during the attack, reflex irritability is retained, and the thumbs, if extended, are at once restored to the flexed position. In spite of all, great tact is sometimes necessary to discover the deception.

If the diagnosis of epilepsy is rendered certain, we must endeavor to ascertain whether the disease is primary or secondary. The diagnosis of a direct affection of the cerebral cortex—cortical or Jacksonian epilepsy—is favored by the fact that the spasmodic seizure is always confined to one limb.

VI. PROGNOSIS.—The prognosis is decidedly grave. Recovery is the exception, rather than the rule. Years may elapse in which no attack occurs, and the disease is regarded as cured, when a relapse proves our error. The hereditary forms of the disease are especially obstinate. It is readily understood that the prognosis is grave in those cases in which the attacks recur with great frequency, are severe and protracted, and associated with mental disturbances.

VII. TREATMENT.—Prophylaxis is indicated with regard to the children of epileptic parents. A mother, who is epileptic or comes of an epileptic or neuropathic family, should not nurse her own child. The children should be nourished with special care, and should avoid bodily and mental strain and sudden mental excitement.

The regimen constitutes an important part of the treatment of epilepsy. Stimulating drinks (alcoholics, tea, and coffee) and articles of food which are difficult of digestion should be interdicted, a daily evacuation from the bowels should be secured, and plethoric individuals may take a cure at Carlsbad, Kissingen, Marienbad, etc. Excesses in Baccho and Venere must be strenuously avoided. Cold baths must be used with caution, since they sometimes prove too stimulating; baths should never be taken except in the company of another individual. Benefit may be derived from lukewarm baths at a temperature of 26° R. (for a half-hour every other day).

Causal conditions must first be taken into consideration as regards treatment—mercury and potassium iodide in syphilis, antihelmintics in helminthiasis, excision of cicatrices with inclosed nerves, removal of

tumors, treatment of ear diseases, scrofula, rickets, trepanation in cases of foreign bodies between the skull and brain.

The treatment of epilepsy as such must be separated from that of the individual attacks.

Among the numerous remedies recommended, the most reliable, in our opinion, are potassium bromide and radix artemisiæ. Potassium must be given in large doses in a large amount of water, and for a long time:

R Potass. bromid.. .... ℥ xiv.
 Aq. destil ....q. s. ad ℥ x.
M. D. S. One tablespoonful t. i. d.

In some of our cases, we feel convinced that the action of the bromide was increased by the addition of artemisia:

R Rad. Artemisiæ............................... ℥ ss.
 Coque cum Aq. dest ....q. s. ad colatur ℥ viij.
 Potass. bromid. .... ℥ xiv.
M. D. S. One tablespoonful t. i. d.

Other bromide salts are also recommended, particularly sodium, ammonium, and lithium bromide; the latter is said to be tolerated much better than the other salts. Erlenmeyer extols the combination of sodium and ammonium bromide in equal proportions. If the drug is given in too large doses or for too long a time, symptoms of poisoning set in—acne, feebleness, mental sloth, weakness of memory, uncertain and tremulous movements, somnolence, and feebleness of the heart. The drug must then be discontinued for a time. P. Rosenbach has shown experimentally, in dogs, that potassium bromide diminishes the irritability of the motor cortical centres.

We have rarely seen any good effects from the use of valerian, belladonna, nitrate of silver, zinc, copper, or gold. According to Albertoni and Unverricht, atropine increases the excitability of the cerebral cortex in animals.

We may make mention of the following remedies, which have been employed in this disease: *a.* Venesection, leeches, cups. *b.* Seton in the neck, issues, derivatives of all kinds. *c.* cold-water cures, salt-water and fresh-water baths. *d.* narcotics: opium, chloroform, chloral hydrate, hyoscyamus, curare, stramonium, strychnine, digitalis. *e.* nervines: asafœtida, castoreum, arsenic, phosphorus, perosmic acid. *f.* Electricity (galvanic current transversely or obliquely through the cerebrum, to the medulla oblongata, or the sympathetic) has secured no positive results hitherto. *g.* Alexander claims to have cured cases by ligature of the vertebral artery.

In the treatment of an epileptic seizure, it is doubtful whether it is advisable to check the attack on the appearance of an aura, since many patients state that they then feel worse than before, and that the next attack is more severe. As a general thing, our treatment during an attack must be confined to placing the patient in a good position, to guarding him against injury, and to prevent acts of violence when he awakens.

Compression of the carotids is irrational, and chloroform narcosis is not devoid of danger; the most rational measure appears to be the inhalation of nitrite of amyl, since this dilates the vessels, and will relieve cerebral anæmia.

Inhalations of chloroform, ether, and nitrite of amyl have been recommended in the treatment of status epilepticus.

2. *Eclampsia.*

I. ETIOLOGY.—The term eclampsia is applied to acute attacks of epileptiform spasms, which have the same genesis as true epileptic attacks, and are produced by irritation of the motor cortical centres of the brain. It may be the result of various causes, such as toxic influences (uræmia and lead poisoning), diseases of the brain and its meninges.

Childhood is especially predisposed to eclampsia, probably on account of the slighter development of the reflex inhibitory apparatus (Soltmann), and in the following remarks we will consider eclampsia infantum alone.

The disease is observed chiefly in nurslings (fifth to twentieth months), and is much rarer in the new-born and in children beyond the age of two years.

Heredity exercises a certain amount of influence, since eclampsia occurs particularly in children whose parents or other relatives are neurotic. Under such circumstances, the exciting cause may be of such a trifling nature that the attack appears to develop spontaneously.

Eclampsia is sometimes the result of violent mental emotion (joy, fright, anger, etc.).

It occurs most frequently in a reflex manner, as the result of peripheral irritations (painful wounds of the skin, eruption of the teeth, inflammations of the buccal cavity, foreign bodies and inflammations of the ear, violent gastric and intestinal catarrh, fæcal stasis, worms, renal and vesical calculi, etc.). Eclampsia is much rarer, under such circumstances, in adults than in children.

It also occurs not infrequently in febrile conditions and infectious diseases in children.

Eclampsia sometimes occurs idiopathically without any demonstrable cause. Rachitic children are attacked not infrequently, perhaps as the result of pressure on the brain by the yielding skull.

II. ANATOMICAL CHANGES of a causal character have not been discovered hitherto. The secondary changes which have been observed are hemorrhages into the meninges, brain, and spinal cord, increase of the cerebro-spinal fluid, etc.

III. SYMPTOMS.—The symptoms develop suddenly, for example, immediately after a fright, or they are preceded by prodromata. The latter consist of changed disposition (restlessness, terror, a tendency to cry), restless sleep, disturbances of appetite and digestion. Slight twitchings in individual groups of muscles often occur during sleep; the children lie with half-closed eyes, grit the teeth now and then, laugh in their sleep, pass urine and fæces in bed, contrary to their usual habit, etc.

The eclamptic seizure itself is exactly like an epileptic attack. It may last from a few minutes to several hours. A status eclampticus sometimes develops, *i. e*, the child does not recover consciousness completely before another attack begins. In such cases the bodily temperature may rise very high, while it is unchanged in shorter attacks.

Sometimes a single attack alone occurs, in other cases the attacks

recur at longer or shorter intervals, or with each eruption of a new tooth.

A fatal termination may result from suffocation in consequence of closure of the vocal cords, or disturbances of the respiratory apparatus, or collapse. Or complete recovery takes place. Slight pareses may be left over for some time; permanent paralyses must be attributed to hemorrhage and lesions of the brain. Fractures and dislocations and breaking of the teeth have been observed as the result of the violence of the muscular contractions. Eclampsia is sometimes followed by epilepsy.

IV. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is easy; the prognosis is always serious.

Children must be protected against sudden mental excitement, and must not be nursed by mothers who come of neuropathic families.

In combating the individual attack, the child must be relieved of all tight clothing, and placed in a large, slightly darkened, airy room, with the head low; it should be given a lukewarm bath (28° R.), with cold douche to the head, and an enema of vinegar or salt given. A mustard draught may also be applied to the breast or calves. In status eclampticus, we may employ inhalations of chloroform, or amyl nitrite and enemata of chloral. After the attack has subsided, causal treatment must be inaugurated, or, if there are no causal indications, we may order potassium bromide or other bromides, in order to diminish the irritability of the cerebral cortex.

3. *Tetanus.*

I. ETIOLOGY.—Tetanus manifests itself by tonic muscular spasms which are interrupted now and then by clonic twitchings, and by increased reflex excitability, consciousness remaining entirely intact.

It is divided into four varieties, viz.; idiopathic, traumatic, rheumatic, and toxic tetanus.

The *traumatic* form is the most frequent. It may be the result of operation wounds or accidental injuries. Among the latter, the most dangerous are contused and torn wounds, wounds containing foreign bodies, and injuries of nerves and tendons. Experience also teaches that tetanus is apt to arise from wounds of the fingers, toes, palms of the hands, and soles of feet. In some cases, it does not develop until pyæmic symptoms have made their appearance.

Tetanus may follow a wound at once. Thus, it has been observed immediately after ligature of the crural nerve in amputation of the thigh. In other cases, it develops after the lapse of days and weeks.

In some patients, certain auxiliary factors seem to further the development of the disease. It has been noticed in several campaigns that the vanquished were attacked more frequently than the victors, so that a psychical influence may be suspected. Irritating treatment of wounds and colds also favor its development, and occasionally epidemic influences seem to play a part.

Even the most insignificant wound (piercing the ear, extraction of a tooth, sting of a bee, etc.) may give rise to the disease. Nor is it necessary that the wound be open. Concussion of the spine and head may act as a cause.

It is sometimes the result of internal wounds. Thus, tetanus sometimes follows an abortion, or even a natural delivery (generally from the third to seventh day after delivery). It occurs occasionally during pregnancy. It has also been known to follow the spontaneous removal of an uterine fibroma, rectal ulcerations, etc.

Tetanus neonatorum constitutes a special group. It begins, as a

rule, between the fifth and twelfth days of life, and in the majority of cases is the result of disease of the umbilicus. In other cases, it follows puerperal infection of the new-born, sometimes fæcal stasis. Smith maintains that the majority of cases are the result of the strong backward pressure of the occiput and the mechanical irritation of the medulla oblongata (convulsive centre).

Rheumatic tetanus was formerly regarded as extremely frequent, now it is considered to be a rare disease. According to recent views it is doubtful whether a cold, as such, is capable of producing tetanus, or whether the action of certain lower organisms does not constitute the real cause of disease.

Toxic tetanus is that form which follows thermal or chemical noxa. The former includes certain cases which occurred in the new-born as the result of the use of hot baths by midwives who were accustomed to measure the temperature of the water with the hand, and whose temperature sense was impaired as the result of disease.

Tetanus following poisoning with strychnine, brucine, or picrotoxine is regarded as the prototype of toxic tetanus, but it must be noted that in this form clonic muscular twitchings are much more prominent than in other forms of tetanus.

The term idiopathic tetanus is applied to those cases in which no cause can be discovered.

Leaving tetanus neonatorum out of consideration, the disease is most frequent in middle life, because there is then more liability to traumatism, which is the most common cause of the disease. For this reason, likewise, tetanus is more frequent in the male sex. It is said that individuals of full-blooded constitutions are attacked more frequently by tetanus, but this is not in accordance with our own experience. In the tropics negroes are affected much more often than the whites, but some authors maintain that this is not owing to race peculiarities, but rather to the facts that the negroes are less cleanly and live under less favorable surroundings. Climate exercises a notable effect. The disease is very frequent and sometimes epidemic, in the tropics, particularly during hot spells, or when hot days are followed by cold nights. General hygienic conditions are also important. In poorly ventilated and uncleanly maternity hospitals, particularly in former times, terrible epidemics of tetanus neonatorum have been observed, which ceased as soon as rules of cleanliness were enforced. Similar conditions may also prove destructive in the community at large.

II. SYMPTOMS.—The disease is generally preceded by prodromata. In traumatic tetanus the patients complain of pain in the wound, and the latter sometimes assumes a bad color. The pains sometimes run along the course of a large adjacent nerve, but this does not justify the conclusion that there is an inflammation of the nerve in question.

Many patients are peculiarly restless, excited, sleepless, and anxious. Pain and stiffness soon supervene in the jaws and muscles of the pharynx and back of the neck, finally muscular spasms make their appearance.

In the new-born, the occurrence of tetanus is preceded not infrequently by inflammation of the umbilicus. The children cry often during sleep, are restless, and not infrequently present disturbances of digestion.

The first manifest signs appear in the face, then in the muscles of mastication and of the pharynx, the muscles of the back of the neck, and finally the tetanic symptoms appear in the muscles of the back and

limbs. In general, therefore, the disease spreads from above down-wards.

In some cases, tetanus of the jaw muscles is especially prominent (trismus), so that the slighter implication of the muscles of the back of the neck is overlooked. But our experience is corroborative of that of those authors who deny the existence of trismus pure and simple.

The muscles of the extremities are not infrequently almost entirely unaffected. This is particularly true of the tetanus of adults.

Tetanus of the facial muscles gives rise very early to a peculiar change of facial expression, which by some writers is regarded erro-

FIG. 122.

Facial expression in a case of idiopathic tetanus in a man æt. 28 years.

neously as a prodromal symptom. The forehead is drawn into horizon-tal and, near the glabella, into longitudinal folds, the contours of the masseters appear distinctly under the integument, the alæ nasi are drawn outward and upward, the mouth is widened, the angles of the mouth are drawn downwards and send out diverging folds of skin, the teeth become visible, etc. The features express the most antagonistic feelings. While the upper half of the face has a cheerful though tired expression (diminution in size of the palpebral fissure), the lower part is sad, and the mouth has the expression of one who is sobbing. On ac-count of the uncovering of the teeth, the expression has been compared to that of laughing, risus sardonicus (vide Fig. 122).

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The patients are able to open the mouth very little or not at all, because the masseters draw the jaws very firmly against one another. Infants are unable to retain the nipple between the jaws; they often apply the lips greedily to the nipple, but quickly let go with a loud cry. The jaws oppose such a resistance to passive motion that it would appear as if the lower jaw would be dislocated or fractured rather than the spasm be overcome. The nutrition suffers on account of this condition, and if the disease lasts a long time threatens inanition.

This danger is increased still further by tetanus of the pharynx, which generally appears very early and is often very marked. The patients often suffer the tortures of Tantalus on account of their inability to satisfy their hunger and thirst.

The muscles of the nape are almost always in a state of tetanic contraction, so that the head is drawn forcibly backwards. The bellies of the contracted muscles stand out distinctly beneath the integument, particularly at the sides. Rotatory movements and increased flexion of the head backwards may sometimes be performed with surprising facility.

Among the muscles of the back, the long extensors generally acquire the predominance. The back, and with it the entire body, is so stiff that the patient can be lifted up *in toto* by the back of the head. In dorsal decubitus, the spine is unusually curved anteriorly, so that the fist may often be passed between the back and bed (opisthotonos).

Much rarer are tetanic curvature anteriorly (emprosthotonos), or to one side (pleurothotonos); the body is rarely held perfectly straight (orthotonos).

If the muscles of the thorax and diaphragm are affected, the respiratory movements are interfered with, and the patient falls into a condition of extreme dyspnœa. He becomes cyanotic, breathes irregularly and with difficulty; finally, carbonic-oxide narcosis sets in, and death from suffocation. These dangers increase when, as is not infrequent, the muscles of the larynx undergo tetanic spasms. Disturbances of speech are associated with tetanus of the muscles of the tongue.

Tetanus of the abdominal muscles is shown by retraction of the abdomen, which is sometimes boat-shaped.

The older reports mention priapism and involuntary discharge of semen. In the case shown in Fig. 122, the penis was permanently in a semi-erect condition (tetanus of the ischio-cavernosus and bulbo-cavernosus muscles?).

In tetanus of the muscles of the limbs, the latter are rigid and permanently flexed or extended, and the tense bellies of the muscles appear prominently under the skin.

In very rare cases is there tetanus of cerebral nerves other than the facial, trigeminus, and glosso-pharyngeus, but strabismus has been described in a number of cases, generally as an ominous sign of approaching death.

The character of the tonic contractions is not always alike. In a series of cases, they persist with almost unchanged intensity for hours, days, and even weeks. They disappear only during sleep or chloroform and chloral narcosis, and reappear very quickly on awaking. In other patients, the tetanic contractions occur only in paroxysms. Such attacks generally begin with clonic twitchings, which often produce violent convulsion of the entire body. They may be produced by very slight irritants, so that it is evident that tetanus is the result of increased

reflex excitability. Gently touching or blowing on the skin, a bright light, the slamming of a door, or even the mere idea of an approaching attack may excite the contractions. The number and duration of the attacks also depend on the severity of the disease. Rapid fibrillary twitchings are sometimes seen to run across the tetanically contracted muscles. The vigor of the contractions may be very considerable, and they sometimes result in fracture or dislocation of the bones or breaking of the teeth.

The sensorium is always unaffected, except that delirium sets in not infrequently towards the end of life.

The patients almost always complain of pain in the contracted muscles, and of obstinate insomnia. The majority experience a sensation of unspeakable terror.

The bodily temperature may be unchanged. In other cases, it is subnormal or there are slight, irregular elevations of temperature. There is sometimes a præmortal, hyperpyrexial elevation of temperature (44.7° C. in Wunderlich's case).

It has been shown in criminals that the bodily temperature rises when general tetanic spasms are produced by strychnine-poisoning or faradization of the spinal cord. Nevertheless, the elevation of temperature does not appear to depend on the muscular contractions alone. It is more probable that this is the result of central influences and of the extension of the nervous disturbances to the heat-moderating centres.

The pulse is generally accelerated, sometimes irregular.

The skin is often covered with profuse sweat, probably as the result of disturbances of innervation of the sweat centre in the medulla oblongata. The cutaneous sensibility is generally unchanged; Demme claims to have found diminution of sensibility to temperature and touch. The cutaneous reflexes are often increased. In a case recently under treatment, the patellar reflex was diminished; it is sometimes found to be increased.

There is generally difficulty in the evacuation of urine and fæces. The urine is generally scanty, highly colored, and of slightly increased specific gravity; it often deposits a sediment of urates. It sometimes contains albumin, occasionally sugar; Griesinger discovered casts and an increased amount of indican.

According to Senator, the urea is unchanged or diminished in amount; kreatinin is diminished. In one of my cases, the amount of urea was diminished, that of kreatinin distinctly increased. The albuminuria may be the result of nephritis (produced by rheumatic influences), or of stasis in consequence of the muscular spasms, or of disturbances of central or peripheral innervation of the renal nerves.

Bronchitis, pneumonia, and acute nephritis are described as complications of tetanus.

The duration and course of the disease are extremely variable. Robinson reported a case in which death occurred, in a negro, fifteen minutes after the receipt of the injury. In other cases, the disease lasts days, weeks, even months, and in favorable cases a long time often elapses before the last traces disappear. The patients often complain for a long time of stiffness, weakness, and drawing pains. Pareses and even paralyses have been mentioned as sequelæ.

A fatal termination may be the result of exhaustion, suffocation, or excessive rise of temperature.

The term head tetanus (tetanus hydrophobicus) is applied to a special form of tetanus, of which seventeen cases have been hitherto reported. The disease starts in injuries to the head, often of a very trifling character. This is followed by trismus and paralysis of all the branches of the facial nerve upon the side of the injury. The facial paralysis either precedes or follows the trismus. The electrical irritability of the nerve remains normal. The paralyzed muscles are not infrequently in a condition of contracture. At a later period, spasms of deglutitition and then general tonic spasms make their appearance. The disease generally terminates fatally. Macroscopically, the facial nerve appears to be intact.

III. ANATOMICAL CHANGES.—Rigor mortis generally sets in very rapidly, and is very marked in the bodies of those dead of tetanus. If death was preceded by a rise of temperature, the latter sometimes continues to increase after death. This has been attributed to the coagulation of myosin and the consequent production of heat.

The muscles are often unusually pale. In places, they contain extravasations of blood, and the microscope shows rupture of the muscular tissue in these parts. In traumatic tetanus, the nerve trunks in the neighborhood of the wound are not infrequently reddened and swollen. In some cases, it is said that these changes extend along the nerves to the spinal cord. Froriep described, in two cases, nodular swellings of the nerves (neuritis nodosa) extending as far as the spinal cord. Umbilical arteritis and phlebitis have been observed in several cases of tetanus neonatorum.

Rigid contraction of the heart muscle has been noticed in a few cases; Rosenthal describes extravasations and rupture of fibres in the heart muscle. Marked contraction of the œsophagus and injection of its mucous membrane are also mentioned.

Meningeal hemorrhages are often found and, as in other spasmodic conditions, are the result of circulatory stasis. These insignificant hemorrhages do not constitute the essence of the disease. If they are more extensive, the cerebro-spinal fluid becomes sanguinolent, and is often increased in quantity, probably a post-mortem change. The substance of the brain and spinal cord may present anæmia, hyperæmia, or small extravasations of blood, or all combined. Various microscopical changes in the nervous tissues have been described, but the observations are not reliable.

In our opinion, tetanus is an infectious disease, produced by the action of definite lower organisms, the nature of which is still unknown. Fluegge and Nikolaier have recently cultivated bacteria which, when injected subcutaneously in animals, gave rise to tetanus-like symptoms. As a matter of course, infectious tetanus does not include the toxic form which is the result of purely chemical influences. It is an interesting fact that, in the decomposition of organic matters, substances are produced which possess strychnine-like effects, and the question arises whether the supposed bacteria do not produce tetanus by forming similar chemical agents during their development.

IV. DIAGNOSIS.—The recognition of tetanus and its etiology is generally easy. It is distinguished from spinal meningitis by the fact that, in the latter, trismus is absent and irritative phenomena predominate. In simple masticatory facial spasm the muscles of the back of the neck are unaffected. I recently saw a case of violent, acute muscular rheumatism of the muscles of the back, which gave rise to persistent, severe opisthotonos, but the muscles of mastication and deglutition were entirely intact.

V. PROGNOSIS.—The prognosis is always grave, but the cases run a very variable course, so that some observers lose nearly 100% of their cases, others not more than 50%. It must be remembered that an apparently favorable case sometimes takes a rapidly unfavorable turn.

Unfavorable signs are elevation of temperature, continued insomnia, delirium, and strabismus. The longer the disease lasts and the more the temperature remains within normal limits, the more favorable are the chances.

As a general thing, idiopathic tetanus presents a more favorable prognosis than the traumatic variety; in the latter, the prognosis improves the greater the interval between the injury and the first symptoms.

VI. TREATMENT.—The causal indications must first be taken into consideration. These include cleanliness and careful treatment of wounds, laying open wounds to remove pus or foreign bodies, removal of ligatures from nerves, etc. Nerves have also been incised in the hope of preventing the spread of an inflammatory process along the nerve to the spinal cord.

The following plan appears to be the simplest and most reliable in the treatment of tetanus per se: milk diet, if necessary the administration of nourishment by means of the œsophageal sound or nutritive enemata; if there is constipation, calomel and jalap, āā. gr. v., and chloral hydrate gr. xxx.–lxxv. daily.

We will simply mention the following among the methods of treatment recommended in this disease: *a.* Derivatives to the spine; leeches, cups, alcoholic inunctions, ether spray. *b.* Antiphlogistics: ice bag to the spine, mercury internally and by inunction, potassium iodide. *c.* Narcotics: opium, paraldehyd, strychnine, belladonna, atropine, eserine, curare, coniine, hyoscyamine, potassium bromide, amyl nitrite, tobacco enemata. Morphine and opium should be avoided, since they are capable of producing tetanus. Nor does curare exercise any good effects against tetanus itself; in the most favorable event it merely checks the spasmodic phenomena by paralyzing the terminal nervous apparatus. In addition to chloral hydrate, reliance may be placed upon large doses of the bromides, since they diminish the reflex irritability of the central nervous system. Paraldehyd (ʒ i–ij. daily) may also be employed. *d.* Nervines. *e.* Electricity: constant descending current on the spine, feeble current, one to two hours' duration, two to three times daily, or the anode labile on the individual tetanic muscles, cathode on an indifferent spot. *f.* Nerve stretching has also been tried.

4. *St. Vitus' Dance. Chorea.*

(*Chorea Minor.*)

I. ETIOLOGY.—Chorea is a neurosis which is located, in all probability, in the brain. The disease manifests itself by muscular restlessness and inco-ordinated movements, which occur spontaneously or as associated movements in voluntary muscular actions, and by changes in the psychical sphere.

It occurs chiefly in childhood, particularly at the period of second dentition (sixth and seventh years) and puberty (eleventh to fifteenth years). It is rare in adults, relatively most frequent from the fifteenth to the twenty-fourth years. In some cases it occurs in old age (chorea senilis), mainly as the result of violent mental emotions.

Some authors report cases of congenital chorea, and it is even said that chorea of the fœtus has been observed in pregnant choreic women (?).

Heredity plays an important part in the etiology of chorea. In a number of my cases the children, parents, and other relatives had suffered from the disease. In other families, there are isolated cases of chorea, while other members suffer from hysteria, epilepsy, psychopathy, and nervousness.

In some cases the predisposition to chorea is acquired. Excessive application in school, excitement following the reading of obscene books, malnutrition, onanism, and anæmia may give rise to such a predisposition, so that slight exciting causes are apt to produce the disease.

Among the direct exciting causes may be mentioned : joy, grief, and particularly fright.

In rarer cases it is attributed to injury, for example, a blow on the head or back, etc.

Chorea is sometimes the result of reflex causes.

Borelli reported a case which recovered after the removal of a neuroma from one of the nerves of the arm. Attention has been called recently to the presence of painful points on peripheral nerves, the treatment of which produced rapid improvement. Wood observed hemichora after amputation of the leg. Fæcal stasis and intestinal worms sometimes give rise to chorea in children. This is also true of dentition and defective condition of the teeth. Chorea sometimes disappears rapidly after the relief of phimosis.

This category probably includes the chorea of pregnancy, in which the irritation starts from the inner surface of the uterus. It occurs generally in primiparæ, particularly if they have suffered previously from anæmia, nervousness, or chorea. As a general thing, it does not appear before the end of the second month, but it is not rare in the second half of pregnancy. As a rule, the symptoms are very violent, terminate not infrequently in death, give rise to abortion, and do not cease until delivery. The disease may then terminate quite suddenly. Occasionally the subsequent pregnancies are accompanied by chorea. Lawson Tait reports the case of a woman who suffered from chorea in four successive pregnancies, and died in the fourth pregnancy despite the occurrence of abortion.

Some of the older writers explain the frequent combination of chorea and heart disease as the result of mechanical irritation of the phrenic nerve by the enlarged heart. Others regard capillary emboli of the cerebral arteries as the connecting link between the two conditions.

Numerous personal observations confirm the frequent connection of chorea with heart diseases, particularly valvular lesions (usually mitral insufficiency), more rarely pericarditis and diseases of the heart muscle. French authors have overestimated the frequency of this connection, inasmuch as they attribute every systolic murmur, which occurs so frequently in the anæmic patients, to mitral insufficiency. We confess that, in our opinion, the previously mentioned theory of the reflex origin of chorea in heart disease seems to be very plausible in many cases.

Chorea sometimes follows infectious diseases, viz., pneumonia, typhoid fever, cholera, diphtheria, variola, morbilli, scarlatina, and, above all, acute articular rheumatism. It also occurs, in very rare cases, with the outbreak of the so-called secondary symptoms of syphilis.

The connection of chorea with infectious diseases is entirely obscure.

In children, it sometimes develops as the result of imitation, i. e., when brought into close contact with choreic patients, the children also

begin gradually to perform choreic movements. This explains its en-
demic occurrence in boarding-schools and convents.

The disease appears occasionally as an epidemic. Steiner reports an
epidemic in Prague, which lasted two months (February and March).
This is perhaps the result of climatic influences. At all events, many
writers agree that chorea is most frequent in the autumn and winter,
particularly if there are sudden changes of temperature.

Girls are affected more frequently than boys (in the proportion of
two to one).

II. SYMPTOMS.—The symptoms rarely begin suddenly; this is most
apt to happen after fright and emotional excitement. As a rule, the
disease is preceded for days and weeks by prodromata, consisting of un-
usual irritability, depression, apathy, disinclination to mental and bodily
work, impairment of memory, anorexia, restlessness, pallor, pains in the
spine, limbs, joints, headache, etc.

Gradually the patients attract attention by their awkwardness. The
fingers do not obey promptly in sewing or playing piano, food is spilt in
eating and drinking, there is inability to stand still, etc. So long as
the motor disturbances are not very marked, they are often regarded as
the result of bad habits or ill-behavior. But soon the symptoms become
unmistakable.

Involuntary pronation and supination, flexion and extension of the
fingers, and twitchings of the shoulders are observed in the upper limbs.
If the patient is directed to slowly grasp an object or thread a needle,
etc., all sorts of zigzag movements are performed, and prevent the con-
summation of the desired action (insanity of muscles, folie musculaire,
muscle delirium). In advanced cases, the arms are thrown violently to
and fro, and the patients are incapable of dressing themselves, or attend-
ing to the ordinary duties of life. It is notable that even in advanced
cases the ability to write is sometimes very little affected.

Corresponding movements are often noticeable in the lower limbs.
They may be so violent that the patient is unable to stand, and, if asso-
ciated with similar twitchings in the muscles of the trunk, he is placed
in danger of being thrown out of bed.

If the muscles of the thorax are markedly affected, considerable
dyspnœa may develop, the patients have a cyanotic appearance, and are
in danger of suffocation.

Grimaces are observed in the face. The patients wrinkle the fore-
head involuntarily, roll the eyes, distort the cheeks, and move the tongue
restlessly, so that speech, deglutition, and mastication are interfered
with. Speech is often entirely indistinguishable. The movements of
the jaw muscles may be so violent as to break the teeth, and the tongue
and mucous membrane of the cheeks are not infrequently injured.

The laryngoscope often discloses choreic movements of the vocal
cords.

The bladder, rectum, and heart are always unaffected; it is true that
many writers speak of chorea of the heart, but the existence of this
symptom is justly doubted.

The patients rarely experience a feeling of exhaustion, despite the
fact that the muscular contractions are kept up almost uninterruptedly.
They cease during sleep, but interfere with the act of falling asleep.
The patients often complain that sleep is interrupted by brief, disagreeable
dreams. The muscles also remain quiet during the artificial sleep pro-
duced by chloroform and chloral hydrate.

The electrical irritability of the nerves is generally unchanged, but is said to be increased occasionally in recent cases.

The movements generally do not begin at the same time in all the muscles. They often begin in one limb, generally an arm, then extend to the corresponding forearm, to the limbs on the other side, and also to the face. They are sometimes confined to one limb, or to the limbs of one side (hemi-chorea), very rarely they appear in a crossed form. The left side of the body presents a decided predisposition, and even in general chorea the movements are sometimes more intense on the left side.

The movements are often intensified by mental and bodily excitement. According to Watson, they diminish, and even cease during deep inspirations. Their intensity sometimes presents a lively contrast in the sitting and recumbent positions.

Sensory disturbances are entirely absent in many cases. Great im-

Fig. 123.
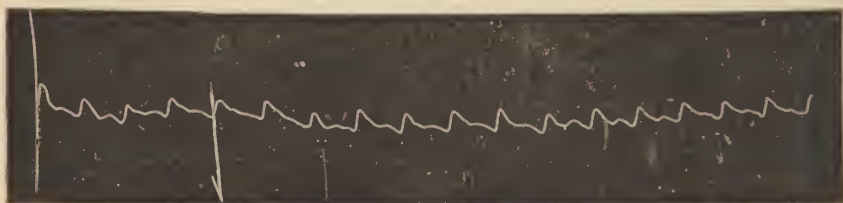
Pulse curve in a girl æt. 12 years, suffering from chorea and mitral insufficiency and stenosis. At the height of the disease.

Fig. 124.
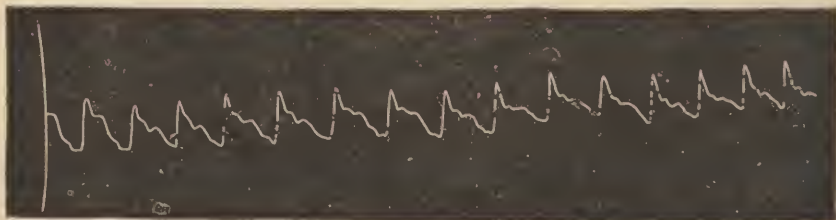
The same during the period of recovery.

portance was formerly attached to painful points on the peripheral nerves, the spinous and transverse processes of the vertebræ, especially of the cervical region, but this symptom is of rare occurrence. Rosenbach and Seiffert found painful points on the peripheral nerves in examination with the electrical current; their electrical treatment was followed by rapidly favorable results.

The mental condition is affected in almost all cases. There is not alone impairment of memory, bad temper, and irritability, but the majority of patients manifest more serious psychical changes, especially causeless laughing and crying and stupid demeanor. In some, imbecility and mania develop. Serious psychopathic conditions are more apt to occur in adults.

The patients often become rapidly pale and emaciated. The bruit de diable is heard over the jugulars, and accidental systolic murmurs are

heard more or less distinctly over the heart valves. There is sometimes slight dilatation of the right ventricle (increased cardiac dulness to the outside of the right border of the sternum), and if the action of the heart is excited, the second pulmonary sound is intensified.

There is often a sort of pseudo-irregularity of the pulse, because it cannot be felt distinctly on the occurrence of choreic movements. The pulse curve in Fig. 123 was taken at the height of the disease, that in Fig. 124 after complete recovery. During the disease the curve is lower and the arterial pressure diminished (more marked elevation of recoil and diminished elevation of elasticity).

The bodily temperature remains unchanged ; in hemichorea there is sometimes a slight elevation of temperature upon the affected side.

Chorea may be attended with not a few complications. There is sometimes painful swelling of the joints, which begins subsequent to the onset of the chorea. Hadden described three cases of right hemichorea with aphasia. Transitory paralyses have also been reported.

The combination of chorea and Basedow's disease has been repeatedly observed. The pupils are often very wide and react slowly to light. E. Remak noticed dilatation of the pupil on the affected side in hemichorea. Stevens mentions the frequent occurrence of hypermetropia, but this is denied by Bull.

Seiffert found diminution of the chlorides in the urine, de Casseres did not verify this observation. According to the latter, the phosphoric acid is generally diminished, the sulphuric acid usually unchanged. Tait found sugar in one case, Leube and Russel report albuminuria in several cases.

Under favorable circumstances the disease lasts from four to twelve weeks. Cases of shorter duration are rare; more frequently they last longer. The symptoms sometimes disappear during intercurrent, acute infectious diseases (measles, scarlatina, diphtheria, pneumonia), but they may reappear after recovery from the latter. The morbid phenomena generally subside gradually, but a sudden termination is sometimes observed if the exciting cause is rapidly removed.

The disease exhibits a decided tendency to relapses. According to Sée, this danger is so much greater if the disease lasts more than sixty-nine days.

In almost all cases it runs a spontaneously favorable course. A fatal termination is rare, and generally occurs quite suddenly after increased intensity of the choreic movements. In rare cases the disease becomes chronic and lasts for life. Paralysis and atrophy have been mentioned as sequelæ. In a few cases the patients remain permanently insane.

III. ANATOMICAL CHANGES.—The anatomical changes of chorea are unknown. Even the site of the disease is uncertain, and the assumption of certain authors that it is located in the corpus striatum or optic thalamus is unfounded.

The anatomical changes which have been described are: meningeal hemorrhages, thickening and calcification of the meninges, increase of the cerebro-spinal fluid, inflammatory proliferation of the neuroglia of the central nervous system and in the blood-vessels, pigmentation and swelling of the ganglion cells, capillary emboli, etc. These lesions are in part doubtful, in part immaterial.

IV. DIAGNOSIS.—The recognition of the disease is easy, although choreiform movements also occur as the result of irritation of the mo-

tor sphere of the cerebrum by meningitis, tubercles, hemorrhage, soft-
ening, tumors, or parasites (symptomatic chorea). The latter conditions
present other symptoms indicative of more serious lesions of the central
nervous system. In hysteria there may also occur violent choreiform
movements, generally associated with hallucinations and disturbances of
consciousness (chorea magna). In post-hemiplegic chorea the choreic
limbs are paralyzed. In athetosis the movements are regular and con-
stant, and there are generally other symptoms of an organic nervous dis-
ease. In multiple sclerosis there is tremor on voluntary movement,
nystagmus, and disturbances of speech.

V. Prognosis is almost always favorable. In children it is better
than in adults. Protracted insomnia is an unfavorable prognostic sign.

VI. Treatment.—Many claim that the disease will run its course
uninfluenced by treatment. But in our opinion the disease will termi-
nate more rapidly under the following simple plan of treatment:

Causal treatment must first be adopted, for example, treatment of
worms, fæcal stasis, defective teeth, phimosis, neuromata, etc. This
also includes the anodal treatment of painful points with the galvanic
current. If the chorea follows rheumatism, we may order salicylic
acid, potassium iodide, propylamin, colchicum, or aconite.

After the causal indications have been met, we should order light,
nutritious food, secure a daily evacuation from the bowels, avoid bodily
and mental strain, and keep the patient as quiet as possible. Every
morning and evening he should take a lukewarm bath (28° R.) lasting
fifteen minutes, and followed by at least half an hour of repose. Inter-
nally the following may be ordered:

R Liq. potass. arsenit.,
    Aq. amygd. amar.............................āā ℥ ss.
M. D. S. Five drops t. i. d. after meals; the daily amount to be in-
creased one drop every three days, until ten drops are taken t. i. d.

If gastric symptoms set in, the arsenic may be given subcutaneously
(gtt. xv. : gtt. xxx. of water, one-fourth to one-half syringeful, once a
day).

A large number of other remedies have been employed, of which the following
are the principal ones: a. purgatives, which, according to the older writers,
should always be given at the beginning of the disease; b, derivatives and ether
spray to the spine; c, nervines: potassium bromide, strychnine, curare, calabar
bean, eserine, hyoscyamine, zinc, copper, silver, gold, etc.; d, narcotics: opium,
morphine, belladonna, chloroform, chloral hydrate, amyl nitrite; e, aniline sul-
phate (gr. ¾ to gr. 1¼ in pill form t. i. d.); f, iron preparations; g, electricity, par-
ticularly the galvanic current transversely or obliquely through the skull. Others
recommended the ascending spinal current; h, gymnastic exercises; i, cold-water
cures.

5. *Præhemiplegic and Posthemiplegic Chorea.*

This term is applied to choreiform movements which either precede or follow
a hemiplegia produced by cerebral disease, generally hemorrhage. The former is
rarer and its significance more serious. The choreiform movements are always
unilateral, corresponding to the distribution of the paralysis, and vary greatly in
intensity.

In præhemiplegic chorea, the symptoms generally appear a few days before
the occurrence of the apoplectic attack, and cease as soon as the paralysis makes
its appearance. Posthemiplegic chorea does not begin, on the other hand, until
the previously paralyzed limbs have regained a certain amount of motor power.
The affected limbs are often contracted and not infrequently anæsthetic. Even

the organs of special sense may take part in the hemianæsthesia. It is probable, therefore, that the lesion is located in that part of the cerebrum at which certain fibres of the pyramid tract in the internal capsule pass between the lenticular nucleus and optic thalamus to the occipital lobe (vide Fig. 104). At times, however, similar symptoms seem to be produced by lesions in other parts, for example, the pons and spinal cord, but, at all events, irritation of some portion of the pyramid tract seems to be necessary.

The treatment is the same as that of the primary disease.

6. *Athetosis.*

The majority of authors look upon athetosis as identical with posthemiplegic chorea, or at least as a modified form of that disease. The symptoms almost always follow an attack of hemiplegia (hemiathetosis), but in some cases they occur in spinal diseases (infantile paralysis, tabes). Bilateral athetosis has been observed in epileptics, insane, and idiots. It is sometimes observed as an independent neurosis; in rare cases it is congenital.

The characteristic symptoms appear in the fingers and toes. The fingers are generally in slow, more rarely in rapid, almost tremulous motion, and are successively flexed, extended, abducted, abducted. The patients are unable to repress the movements entirely, and they continue during sleep. They are sometimes inhibited by firmly grasping the wrist with the fingers of the other hand, or by raising the arm erect. The thumb, index, and little fingers generally are most affected, and the external and internal interossei are chiefly involved. The wrist may also be affected, and not infrequently performs movements opposed to those of the fingers. During sleep the movements diminish or subside almost entirely; they often increase on excitement. Subluxation of the phalangeal joints and malposition of the fingers and hands may develop after protracted duration of the disease. The paralyzed limbs may undergo contracture. The muscles of the affected forearm sometimes undergo hypertrophy, but they may also be unchanged in volume or even atrophied. Hemianæsthesia is occasionally observed. The electrical excitability is unchanged.

Similar symptoms may be manifested by the toes and feet and by the muscles of the face and back of the neck.

The prognosis is unfavorable as regards recovery, but Gowers reported one successful case. Improvement is said to be obtained at times by descending spino-muscular currents, potassium bromide, and arsenic.

7. *Shaking Palsy. Paralysis Agitans.*

(*Parkinson's Disease.*)

I. ETIOLOGY.—Paralysis agitans, which is characterized by trembling movements of the limbs, diminished muscular power, muscular rigidity, slowness of muscular contraction, peculiar position of the body, changed facial expression, and forced movements, is not a frequent disease.

The causes mentioned are: colds, injury, fright and mental excitement, heredity, infectious diseases, alcoholic excesses, gout, excesses in venery, particularly coitus in the standing position. In some cases no cause is demonstrable.

The influence of a cold cannot be denied, because it is sometimes followed at once by the symptoms of the disease. Among traumata, injuries of the nerves are especially to be dreaded. S. Martin recently described a case in which the disease followed an injury of the radial nerve. Westphal saw a case following simple cutaneous burn of the second degree. In not a small number of cases the disease developed immediately after severe fright. The importance of heredity has only been recognized of late years. In some cases, the disease is conveyed as such to the descendants; in others, it alternates with epilepsy, hysteria, and allied conditions. Romberg observed a case after intermittent fever, and a few cases have been known to follow typhoid fever and acute articular rheumatism.

Sex does not appear to possess much influence on the frequency of the disease. It is most common at an advanced age (forty to sixty years), and is very rare before the age of twenty years.

Among six thousand cases of nervous diseases, Beyer observed paralysis agitans thirty-seven times (0.6 per cent).

II. SYMPTOMS.—The symptoms sometimes develop suddenly; in other cases, they appear so gradually that we are unable to determine the beginning of the disease. A sudden onset is especially apt to occur after fright.

Prodromata are sometimes experienced, such as neuralgiform pains, paræsthesiæ, a feeling of dulness, vertigo, etc.

The first manifest symptom is the tremor. As a rule, it first affects the muscles of the fingers and hand, and later extends to the arms and lower limbs. It begins generally in the right arm, then the right leg, then the left arm, and finally the left leg. In some cases, it is confined to one limb or is distributed in a hemiplegic manner, or affects the arm on one side, the leg on the other, or it may appear in paraplegic distri-

FIG. 125.

Writing position of the hand in paralysis agitans. After Ordenstein.

bution. The muscles of the head and face are generally, though not always, unaffected; in some cases even the muscles of the tongue take part. Oscillating movements may be conveyed from the limbs to the head.

The trembling movements generally continue uniformly during repose and motion; at all events, they do not increase during voluntary movements. On the contrary, the patients are often able to inhibit them voluntarily for a short time. They often increase in intensity during excitement. At first, they cease during sleep, but if the disease has lasted for a long time, they continue, though with diminished intensity, during sleep. The majority of patients complain that the tremor interferes with falling asleep.

At first the muscular excursions are often very slight, but they gradually increase, and often pass from mere tremor into violent shaking. Under such circumstances, the floor, chair, and bed may be vigorously shaken. Writing and other delicate manipulations are interfered with, and finally rendered impossible; indeed some of the patients must be

clothed and fed for years. The tremor sometimes presents remissions and exacerbations.

Charcot has shown that in rare cases this important symptom is absent while all the others may be present. Moreover, the disease may begin with muscular paresis, and the tremor may not develop until a later period.

The trembling movements are soon followed by a peculiar position of the limbs, trunk, and head as the result of paresis and rigidity of certain muscles.

FIG. 126.

FIG. 127.

Position of the fingers in paralysis agitans, similar to that in arthritis deformans.

The diminution of muscular power has been shown by the dynamometer in a number of cases. In others, the muscles retain their normal power, but rapidly become exhausted. In the arms, the extensors are affected earliest and most markedly, while the flexors are more apt to suffer from rigidity and spasms. This gives rise to very peculiar changes in the position of the fingers and hands. In many cases the thumb and fingers are simply approximated, and assume the writing position (vide Fig. 125). This position, together with the constant movements of the thumb and index finger, produces an appearance as if the patient were continually rolling a ball between the thumb and finger. In other cases, the position of the fingers resembles that of

arthritis deformans (vide Figs. 126 and 127), *i. e.*, the first phalanges are flexed, the middle ones hyperextended, and the terminal phalanges flexed. The forearm is generally flexed on the arm, the elbow is somewhat removed from the thorax. In exceptional cases, the arms hang down alongside the thorax.

The knees are sometimes approximated so closely that they cross each other in walking. The constant friction of the knees may give rise to erythema, ulceration, and gangrene. The feet are in pes varoequinus position, and the toes present deformities similar to those noticed in the fingers.

Fig. 128.

Position of the body (bent over forwards) in paralysis agitans. After Charcot.

As a rule, the head is bent over forwards, more rarely backwards. On account of the muscular rigidity, it offers resistance to passive motion.

The trunk is also bent over forward, as if the patient were in constant danger of tumbling head over heels (vide Fig. 128).

The affected limbs generally present no change in color or volume. In rare cases they are emaciated, and in one case hypertrophy of the muscles has been described. In one of my cases in which the tremor was confined to the right upper limb, the arm was constantly reddened, and was 2.5° to 3.5° C. warmer than the opposite limb. Grasset and Apolinari observed elevation of the cutaneous temperature amounting to

3.2° C., while voluntary movements of the limb in healthy persons produced a rise of temperature of only 1° to 2° C.

The electrical excitability in the affected limbs is unchanged.

Cutaneous sensibility is almost always intact. Hadden recently described increase of all the tendon reflexes.

Painful pressure points are sometimes found on remote parts: the skull, spine, shoulder, and peripheral nerves.

A very important symptom is the change of facial expression. The features become peculiarly stiff, lifeless, mask-like; the play of expression ceases, and the patient acquires a stupid expression. Speech becomes slow and monotonous, and is often high pitched, almost tearful. If the muscles of the lips and tongue take part in the trembling movements, speech is also interfered with mechanically. Many patients keep the mouth open constantly, and the saliva often dribbles uninterruptedly. Even when the mouth is kept closed, the buccal cavity often contains very large amounts of saliva. Whether this is the result of increase of the salivary secretion is doubtful, since it may depend upon the difficulty in deglutition, which increases more and more in many patients.

Debove recently called attention to peculiar disturbances of vision. To a certain extent, the patients have lost power over the movements of the eyes. In reading, they are unable to pass quickly from the end of one line to the beginning of the next, and if the reading matter consists of adjacent columns, the eye passes from one column to the next one on the same line. The ocular muscles do not take part in the oscillating movements.

The patients walk bent over forward, the gait is festinating, becomes quicker and quicker (propulsion); there is inability to stand still suddenly, i. e., the patients perform a sort of forced movement, and often fall to the ground, if not assisted. It is also very difficult for the patient to turn around suddenly. Some patients, if their garments are pulled backwards, begin to walk backwards, the movements become more and more rapid, and finally they fall (retropulsion).

The causes of these forced movements are unknown. They have been attributed to the changed position of the body and the consequent dislocation of the centre of gravity of the body, so that the patients are forced, so to speak, to run after their centre of gravity. This explanation is unsatisfactory.

The temperature and pulse are unchanged, but many patients experience a subjective sensation of increased heat.

The sensorium is often undisturbed during the entire course of the disease. But many patients complain of an annoying feeling of restlessness, and in some cases apathy, imbecility, and well-marked forms of insanity appear towards the end of life.

The bladder and rectum are unaffected, except that incontinence of urine and fæces may occur towards the end of life as the result of increasing marasmus. Obstinate constipation has been noticed in a number of cases.

The disease generally lasts many years, sometimes even more than thirty. It exhibits a constant tendency to grow worse, and intermissions or remissions are rare. Apoplectiform attacks (without anatomical basis) sometimes occur as in multiple sclerosis or, in rarer cases, in locomotor ataxia. Death is the result of intercurrent diseases, particularly bronchitis and pneumonia, or of increasing exhaustion. Severe bed-sores

and their sequelæ are sometimes observed. The tremor may subside
completely for a few days before death.

III. ANATOMICAL CHANGES.—Anatomical changes in paralysis agi-
tans are unknown, so that the disease must be regarded as functional.

Atrophy, accumulation of pigment in the ganglion cells, and arterio-sclerotic
changes have been found in the brain and spinal cord, but these appearances are
the result of senile processes. Even small patches of sclerosis in old people are
devoid of significance. The older writers often mistook paralysis agitans for
multiple sclerosis. Luys claims recently to have discovered hypertrophy of the
ganglion cells in the pons Varolii, and explains the disease as a "hypersecretion
of motor impulse " (!).

The disease is located by some in the corpora quadrigemina, by others in the
pons and medulla oblongata. R. Remak distinguishes a cerebral and spinal
form; the disturbances in walking are said to be characteristic of the former.

IV. DIAGNOSIS.—The diagnosis is easy if we take into consideration
the entire complex of symptoms. It is distinguished from simple tremor
by the facts that in the latter the oscillating movements are slighter, that
other symptoms are absent, and that, in mercurial, lead, and alcoholic
tremor, evident injurious factors are demonstrable. It cannot be mis-
taken for multiple sclerosis if we remember that in the latter tremor
occurs only on voluntary motion, nystagmus is present, and the symp-
toms almost always begin in the lower limbs. In chorea, the movements
are irregular, cease at times, become greater during voluntary move-
ments, and the excursions are larger but slower.

V. PROGNOSIS.—It is impossible to check the constant advance of
the disease. Life is not put in immediate danger by the disease, and
many patients live for years after its inception.

VI. TREATMENT.—Treatment must be confined, in general, to con-
stitutional measures. Trial may be made of Fowler's solution (one part
to two of water, one-quarter to one-half syringeful subcutaneously), but
this has produced very little effect in our cases.

Various authors have recommended narcotics, nervines, iron, baths, cold-
water cures, electricity (generally the galvanic current to the head, cervical
cord, cervical sympathetic or limbs, according to circumstances), and nerve
stretching.

8. *Tremor.*

I. SYMPTOMS.—Tremor is manifested by brief, rapidly following
oscillatory muscular movements, which are independent of the will or
volitional movement. In some cases, it is confined to individual mus-
cles or groups of muscles; in others, it affects a whole limb, or is dis-
tributed in an hemiplegic or paraplegic fashion, or extends to almost
the entire muscular system.

The muscles of the fingers and hands are affected most frequently,
sometimes more markedly on the right side. In other cases, the mus-
cles of the head and neck are chiefly affected, so that the head is con-
stantly wagging to and fro. In the face, the muscles supplied by the
facial nerve are most apt to be affected, sometimes the muscles of the
tongue, so that articulation is interfered with, rarely the ocular muscles
(nystagmus). If there is violent tremor of the lower limbs, the knees
may be rubbed so vigorously against one another as to give rise to in-
flammations of the skin, excoriations, and even gangrene.

The tremor often subsides if the tremulous limbs are efficiently sup-

ported, and it also ceases during sleep. It may be repressed temporarily by an effort of the will by many, though not by all patients. It is increased, in some cases, on attempting to execute a movement.

Tremor does not give rise to any danger, but is attended with many annoyances, and interferes with various occupations. The condition is usually very obstinate.

II. ETIOLOGY.—The causes of tremor are manifold. It may be the result of mental excitement, such as anger, joy, sorrow, or fright. In other cases, it follows bodily strain, such as lifting heavy loads, standing on the tips of the toes, etc. It is sometimes the result of toxic influences, such as excessive use of coffee, tea, tobacco, working in lead or mercury, alcoholic excesses, chronic opium eating. Closely related to, often perhaps identical with the toxic forms of tremor, is that form which follows conditions of exhaustion (nervous and neurasthenic individuals, vital losses, onanism, prolonged lactation, convalescence from serious diseases, etc.). According to Charcot, tremor is a constant symptom of Basedow's disease. A well-known form is senile tremor. The symptom may also be produced by thermal influences and by injury.

The genesis of tremor evidently varies according to the causation. In some cases, it depends, as was shown by Frensberg's experiments, upon changes in the circulatory apparatus, such as are produced by psychical and thermal influences. In other cases, it is a phenomenon of exhaustion, in still others of disturbances of co-ordination between certain groups of muscles and their antagonists. The finer processes in the nervous system upon which tremor depends are unknown.

III. TREATMENT.—As a matter of course, the first desideratum is to remove the cause. In the next place, benefit may be expected in many cases from the application of electricity; if the causes are central, galvanization of the brain or cord, otherwise galvanization of the peripheral nerves and faradization of individual muscles. Good results have been obtained from gymnastic exercises, or the application of orthopædic apparatus.

In conditions of weakness, we may order iron, quinine, nourishing diet, cold-water cures, a trip to the country; in nervousness and neurasthenia, valerian, strychnine, veratrine, potassium bromide, and other nervines.

Eulenberg regards Fowler's solution as, in a certain sense, specific, and Oulmont obtained good effects from hyoscyamine (gr. $\frac{1}{20}$ to $\frac{1}{6}$ daily). Gnéneau de Mussy succeeded in curing mercurial tremor very rapidly with zinc phosphide (gr. $\frac{1}{12}$ t. i. d. in pill form).

9. *Dizziness. Vertigo.*

1. Dizziness is the term applied to an interference with the consciousness of the equilibrium of the body in space. The sensation of dizziness has been located in the cerebellum, but this is not strictly proven. The retention of the equilibrium of the body is influenced to a great extent by various sensory impressions, so that, when the latter are disturbed, dizziness often makes its appearance.

2. Dizziness is a symptom of various conditions. In rare cases, it is, to a certain extent, an independent condition, such as occurs in old age, probably as the result of vascular changes in the brain. Perhaps such

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changes give rise to vertigo in the majority of cases. The symptom is rare in childhood.

It is often the result of intracranial changes, such as meningitis, hydrocephalus, tumors, encephalorrhagia, encephalitis, hyperæmia, anæmia, etc. The symptom is apt to be especially severe if the lesion is situated in the posterior fossa near the cerebellum.

It is often the result of reflex causes, such as gastric affections (vertigo a stomacho læso), coprostasis, foreign bodies, worms, or digital examination of the anus. It sometimes follows the ingestion of certain articles of food. Erlenmeyer reported a case which was cured by the relief of an urethral stricture. Soltmann observed it in a boy as the result of retention of the testicle in the inguinal canal. Vertigo is also observed in heart diseases, but this is probably the result of anæmia or hyperæmia of the brain. Charcot described vertigo following disease of the larynx.

Dizziness may be associated with diseases of the organs of special sense. It is a very common symptom in paralysis of the ocular muscles, and is equally frequent in various diseases of the ear (foreign bodies in the external auditory meatus, accumulation of wax, inflammations of all kinds). Some individuals suffer from violent vertigo when air is blown into the Eustachian tube. Diseases of the labyrinth, particularly the semicircular canals, are accompanied by very intense attacks of vertigo (Ménière's disease).

Vertigo is sometimes associated with general anæmia or plethora. It may also be the result of toxic influences (alcohol, opium, nicotine, ether, etc.). Closely allied is that form which is the result of an infection; for example, Kohn reported two cases of intermittent vertigo which were relieved by the administration of quinine.

Vertigo may also be produced by the application of electrical currents to the skull; in many it appears even when the electrical current is applied at a distance from the skull, for example, in the neck.

It is not infrequently the result of impaired ability of determining one's position in space. Thus, gazing at a flowing stream, turning around in a circle, swinging, sailing, climbing high mountains and towers, etc., may produce vertigo.

Vertigo may also be occasioned by a psychopathy, for example, in "fear of places."

3. Dizziness may exist alone or may be followed by other functional disturbances of the brain: vomiting, disturbances of special sense, loss of consciousness, spasms, etc. The majority of patients are dizzy during the day, particularly in the erect position; more rarely the sensation occurs only during dreams (nocturnal vertigo), or is increased in the horizontal position. Sometimes the patient himself appears to revolve, sometimes surrounding objects appear to revolve around the patient. The movement may seem to be rotatory, horizontal or vertical. In some individuals the dizziness occurs only in the morning when the stomach is empty, while in others it increases after meals. The symptom is very annoying, makes the patient anxious, and interferes with freedom of action.

4. In making a diagnosis, the cause should always be sought for, since upon this depend the prognosis and treatment.

10. *Catalepsy.*

1. Catalepsy is characterized by attacks of more or less complete loss of consciousness, during which the limbs permanently maintain any position which may be assumed, but may be passively brought into another position, in which they will then remain.

The symptoms sometimes develop suddenly, but are generally preceded by prodromata: changed mood, feeling of pressure in the head, vertigo, palpitation of the heart, yawning, eructations, etc.

When the attack occurs, the patient remains as if petrified in the position in which he happened to be at the time. The eyes are fixed, the features are unchanged; if the arm has been raised, it remains in that position, etc. The patient is unable to change his position, when asked, even if he understands the request. The muscles appear tense, but passive movements may be performed with very little force, and the limbs may thus be brought into the strangest positions. The readiness with which the limbs may be brought into any position desired, and the persistency with which they remain in such positions, are known as waxy flexibility of the muscles.

This condition generally extends over the entire muscular system, sometimes it begins in one limb, and then spreads to the rest of the body. Rosenthal found increase of the direct and indirect electrical excitability of the muscles during the attacks. Benedikt, on the other hand, observed increase of indirect galvanic excitability, diminution of direct and indirect faradic excitability. Onimus states that muscular contractions are produced more readily by the direct than by the indirect application of galvanism.

The involuntary movements remain unchanged. While the patients are often unable to swallow voluntarily, deglutition is unobstructed if the bolus is placed behind the root of the tongue. Respiration and the action of the heart are unchanged or, at the most, slowed.

Consciousness may be entirely abolished, or only more or less impaired. In the former event, the patients remain ignorant of what happened during the attack. They keep the eyes closed, and awake—often with a loud sigh—at the end of the attack as if from a deep sleep.

In some cases, reflex excitability is entirely abolished, the pupils are dilated, and do not react to light. In milder cases, reflex movements may be produced, but the patients often state that they did not feel the cutaneous irritants.

The skin is sometimes very pale and its temperature very low, but the movements of the heart remain audible, thus distinguishing the condition from death.

An attack may last from a few minutes to hours and even days. In some cases, only a single attack develops; in others, they recur for months and years.

If the condition is protracted, there is danger of inanition, so that the administration of nourishment through the œsophageal sound becomes necessary.

2. Catalepsy is one of the rarer diseases. In some cases, it is a complication of other neuroses, such as hysteria, chorea, melancholia, and various other forms of insanity. It has also been observed in meningitis, encephalitis, and encephalomalacia. It is sometimes the result of great mental excitement, particularly fright, and of religious sentimentality. Some patients mention injury as a cause, for example, a slap in the face. It has been observed in typhoid and intermittent fever—in the latter affection as periodical attacks. Cataleptic attacks may also occur under the influence of chloroform or ether.

All of my patients were in a condition of profound anæmia. The disease occurs even in childhood, and is most frequent during the period of puberty; in women it is sometimes observed at the beginning of pregnancy.

3. The diagnosis is easy; the prognosis depends on the primary affection. The treatment must be directed chiefly against the general condition of the patient, since very little, except the application of cutaneous irritants, can be done to relieve the individual attack.

4. The pathogenesis is entirely unknown.

11. *Hysteria.*

I. ETIOLOGY.—Hysteria is the term applied to a central neurosis in which the brain, cord, and sympathetic may take part, and which is char-

acterized chiefly by increased excitability of these parts. Conditions of intensified or diminished function alternate or coexist in manifold nerve tracts.

The disease occurs almost exclusively in women, and is rare in men.

The symptoms generally become prominent at the period of puberty (fifteenth to twenty-fifth year), but in the majority of cases distinct prodromata are noticeable during childhood.   Indeed, in my experience, well-developed hysteria is not at all uncommon in children.

Like almost all neuroses, hysteria is an exquisitely hereditary disease, which is either transmitted directly or alternates with epilepsy, insanity, and allied conditions.   The hereditary influence of the mother is especially marked; but fathers who are themselves free from nervousness, but who come of a neurotic family, may convey hysteria to their female offspring.

In some cases, hysteria is not hereditary, but the result of a congenital predisposition.   Thus it is said that hysteria is especially apt to develop in those children whose parents married late in life, or suffered from phthisis, or were weak and feeble at the time of conception.

There may also be an acquired predisposition to hysteria.   This is observed in individuals who, since childhood, have been improperly educated, mentally and physically.   We refer to improper diet, insufficient exercise, exciting and obscene stories and books, stimulation of false pride, want of intercourse with other children, too early entrance into society, etc.

The tendency to hysteria may also be acquired by adults.   This may develop after severe and protracted diseases, for example, typhoid fever, phthisis, excessive lactation, losses of blood, chlorosis.

Mental excitement, resulting from worry, care, disappointed hopes, exercises a great influence on the development of hysteria.

A remarkably large number of cases are reflex in their origin, the result of diseases of the peripheral organs.   Some writers believe that this form constitutes almost the only form of the disease.   The peripheral irritation often starts from diseases of the female sexual apparatus, especially the uterus.   I have seen a number of cases in individuals suffering from floating kidney.   This is also true of gastric affections, particularly painful cicatrices or round ulcers and other chronic organic diseases.

Certain diseases of the female sexual organs are particularly apt to be associated with hysteria. Among the diseases of the uterus may be mentioned flexions, versions, and ulcerations, while tumors rarely give rise to hysterical symptoms.   Hysteria is also rare in large cystoid degenerations of the ovaries, but is much more frequent in dermoid cysts, and acute and chronic inflammatory conditions of the ovaries.

Hysteria is not infrequently a disease of married life.   Women become hysterical if their husbands are impotent, if the marriage is unhappy, if they remain sterile, or cohabit too frequently.   Sexual continence is also said to act as a cause of the disease.

Imitation plays an important part in some cases.   In repeated instances, the disease has developed in a previously healthy individual as the result of witnessing an hysterical attack.   This explains the epidemic and endemic occurrence of hysteria in schools, convents, etc.

In the majority of cases, several causes combine to give rise to the disease.

II. ANATOMICAL CHANGES.—Death rarely occurs immediately after an hysterical attack, so that few autopsies have been obtained. No lesions have been discovered hitherto, so that the disease must be regarded as the result of molecular changes.

III. SYMPTOMS.—The symptoms rarely begin suddenly; they generally begin with slight manifestations, and gradually grow more and more intense. Motor, sensory, vaso-motor, trophic, secretory, and psychical disturbances make their appearance, and often alternate with one another, or local diseases of individual organs are produced. On account of the Protean character of the clinical history, we must confine ourselves to an enumeration of the individual possible symptoms.

Among the motor disturbances, paralyses are remarkably frequent. They are confined to individual groups of muscles, or appear as monoplegia, paraplegia, or hemiplegia. Crossed paralysis or paralysis of all the limbs is rarer. Hysterical hemiplegia is associated not infrequently with hemianæsthesia of the paralyzed side—a fact which should at once arouse the suspicion of hysteria, especially if the face and tongue are not paralyzed. The hysterical origin of the paralysis is often evident from the fact that the paralysis varies very greatly within a short period. The paralysis sometimes disappears as suddenly as if blown away, and then again it may reappear as unexpectedly. Violent mental excitement or hystero-epileptic seizures sometimes give rise to paralysis, sometimes they cause its disappearance. Nothing definite can be predicted concerning the duration of the paralysis. It may last a day, sometimes it continues for months, years, even for a lifetime.

Paralyses of individual groups of muscles and monoplegias are more frequent in the upper than in the lower limbs, while paraplegia is more frequent in the latter. Hemiplegias are more common on the left side.

Even after the paralysis has lasted for a long time, it is not followed by atrophy or changes in electrical excitability. At the most, there is slight atrophy from disuse and trifling diminution of electrical excitability.

The facial and hypoglossal nerves possess a certain degree of immunity. Hysterical paralyses of the ocular muscles are also rare; ptosis is relatively frequent.

If the muscular tissue of the pharynx and œsophagus is paralyzed, disturbances of deglutition are produced which may give rise to danger of inanition, unless nourishment is introduced through the œsophageal sound. The unobstructed introduction of the sound is evidence that we have to deal with a paralytic condition.

Not infrequently there is paralysis of some of the laryngeal muscles, generally the internal thyro-arytænoid and posterior crico-arytænoid. The patients are hoarse and aphonic, and, in paralysis of the last-mentioned muscles, suffer from inspiratory dyspnœa, which may reach a dangerous height, and render tracheotomy necessary. Hysterical paralyses of the laryngeal muscles often enable us to observe the effects of unusual stimuli and of moral suasion on hysterical phenomena. Patients who are ignorant of the action of the faradic current may be cured at once upon touching them with the electrode, and the laryngoscope may also show that the paralysis has disappeared. In the majority of cases, such results are not permanent.

Muscular contractures also are often observed in hysteria. They may develop gradually or suddenly, may or may not be preceded by paralysis, may be of short duration, or last for years. They are most frequently-

the results of mental excitement and hystero-epileptic attacks, more rarely of injury, or appear to develop spontaneously. By means of certain manipulations, Charcot was able to produce contractures at will in hypnotized hysterical individuals. Contractures in the arms are usually in the flexed position, those in the lower limbs in extension or hyperextension. They grow less intense, but do not disappear during sleep; they may subside entirely during chloroform narcosis. If contracture has lasted for a long time, the affected muscles undergo atrophy. Joint deformities may also be produced as the result of pressure, so that the use of the limb may be impaired even after relief of the contracture. Charcot believes that the contracture may provoke sclerotic changes in the lateral columns of the cord, and that this explains the exaggeration of the tendon reflexes which is sometimes observed. Great mental excitement and unusual impressions of all kinds may cause rapid disappearance of the contracture, but relapses are the rule rather than the exception.

Many hysterical patients present obstinate tremor, but it is often difficult to decide whether this is the effect of hysteria, or of co-existing anæmia and weakness.

Tonic and clonic muscular spasms form an important symptom. They may appear as tic convulsif or as paroxysmal twitchings in individual groups of muscles in the limbs. Almost all of the muscles of the body sometimes take part in the spasm, but consciousness is often retained. Cases also occur with a distinct epileptic character (hystero-epilepsy). The patients often experience a sort of aura, a sensation of something rising in the abdomen, œsophageal spasm, pallor, occasionally hallucinations, and delirium.

Charcot divides hystero-epileptic attacks into distinct periods. They begin with epileptiform symptoms. Then follow peculiar distortions and changes of position in the trunk and limbs (clownism), marked opisthotonos, lifting of some of the limbs, statuesque poses, etc. Then follows the stage of passionate attitudes, in which the features express fright, joy, or lust; the patients often make use of cynical and erotic expressions. In some cases, the attack ceases abruptly; in others, it gradually subsides. The patients know nothing of what has happened. They often fall into a profound sleep lasting several hours, from which they awaken greatly refreshed.

These hysterical convulsions may occur spontaneously or are provoked by mental emotions. Imitation also plays an important part in this particular. In many patients, convulsions may be produced by pressure on certain hyperæsthetic points in different parts of the body. According to Charcot, they are especially apt to occur after pressure on the ovarian region, particularly on the left side. In other cases, pressure on such points diminishes the severity of the attack or inhibits it altogether.

The attacks sometimes return in such rapid succession as to give rise to a sort of status hystero-epilepticus. Under such circumstances, Wunderlich observed increase of the temperature of the body to 43° C., and death.

Hystero-epilepsy is closely allied to the laughing, crying, and shouting spasms, from which these patients suffer not infrequently. Catalepsy may also occur in hysterical individuals.

Sensory disturbances are as common as motor symptoms, and may or may not be associated with the latter. Anæsthesia, paræsthesia, and hy-

peræsthesia alternate with one another or are present at the same time. Sensory disturbances are probably the result of hysteria, if they vary considerably within a short period and if their distribution does not follow definite nerve tracts.

Profound anæsthesia is most frequent on the soles of the feet and palms of the hand. In other cases, it involves a certain nerve, a limb, one-half or the entire body. Hysterical hemianæsthesia is often associated with hemiplegia; this is supposed to be occasioned by a functional disturbance of the posterior part of the internal capsule (Fig. 104, *Sens.*).

All the modes of sensation or only individual ones may be diminished or abolished. In hemianæsthesia, there is not infrequently complete anæsthesia, which involves the skin, fasciæ, joints, muscles, and organs of special sense. The mucous membranes may also be anæsthetic.

The anæsthetic parts are often pale and cold, and discharge an extremely small amount of blood when pricked with a pin (complication with vaso-motor disturbances).

Among the symptoms of hyperæsthesia, neuralgias are especially frequent and distressing. They affect the trigeminal or occipital nerves, the intercostal or lumbar nerves, or they appear as mastodynia and coccygodynia. Their location sometimes changes in a comparatively short time.

Many hysterical individuals complain of headache, which may be diffuse or unilateral. In two of my cases, the headache was followed by pronounced aphasia, which disappeared in a few hours. Complaint is often made of a boring pain high up on the vertex (clavus hystericus), others experience a sensation of the application of a cold body (ovum hystericum). Symptoms of spinal irritation are not uncommon.

Special attention is merited by the painful or hysterical pressure points. They are found upon the skull, spine, ribs, certain parts of the limbs, or other localities. French authors emphasize the frequency of ovarian tenderness (situated immediately above Poupart's ligament). Left ovarian tenderness (ovarialgia) is especially frequent. It is said that this tenderness is found on that side of the body on which other unilateral hysterical symptoms are found, and that pressure upon the ovary will produce such symptoms or cause them to disappear. German writers do not admit the frequency of ovarialgia, and in many cases it is doubtful whether it is the result of an affection of the ovary, since·painful points are found not infrequently upon the abdominal walls, and may give rise to error. Such painful points are sometimes very extensive, and may arouse the suspicion of peritonitis.

Articular neuroses may also be noticed (vide page 62). In many patients, increased irritability of the smooth muscular fibres of the skin is shown by the almost constant goose skin (cutis anserina hysterica).

The patients often complain of paræsthesiæ: a feeling of cold or heat, prickling, etc. These symptoms sometimes occur in attacks of short duration, in other cases they continue for a long time. They may or may not be associated with vaso-motor changes (pallor of the skin, lower cutaneous temperature, diminished turgor of the skin). Among the vaso-motor and secretory changes may be mentioned cutaneous hemorrhages and sweats (local, unilateral), etc.

Ocular symptoms are often noticeable in hysteria. Ptosis and other forms of ocular paralyses have been previously mentioned. Galezowsky has observed hysterical contracture of the ocular muscles with secondary

diplopia. Foerster applied the term copiopia hysterica to a not infrequent group of symptoms, produced by hyperæsthesia of the optic nerve and orbital branch of the fifth nerve. The patients complain of pains around the globe, which often cease at night, and reappear in the day; they are increased by reading, sewing, and straining the eyes. As a rule, no change in the eye is noticeable on inspection, except that in exceptional cases the pupil is destitute of reaction; visual power is generally unimpaired.

Many patients complain of unusual sensitiveness of the retina, and are distressed to an unusual degree by bright light, especially by red.

Amblyopia and amaurosis are sometimes observed, either on one or both sides; in the former event, hemianæsthesia is often present. This is associated with diminution of the field of vision and disturbances of the color sense. According to Galezowsky, hemianopsia also occurs, but Leber does not consider his cases convincing. Ophthalmoscopic appearances are generally negative, but it is said that hysteria sometimes gives rise to inflammation and atrophy of the optic nerve.

Many patients complain of spots before the eyes, flashes of light, etc. During hysterical attacks, there may be visual hallucinations in which rats, snakes, and small animals play an important part.

Some patients enjoy unusual powers of hearing. They hear sounds which are inaudible to healthy individuals, or are annoyed to an unusual extent by ordinary sounds. In other cases, there is partial or complete loss of hearing on one or both sides—the former generally in hemianæsthesia. Under such circumstances, there is abolition of sensibility in the external auditory canal, upon the membrana tympani, and the mucous membrane of the tympanic cavity. Some patients complain of subjective auditory sensations, and hallucinations of hearing may occur in hysterical seizures.

Smell and taste may suffer in a similar manner. The patients detect by their odor objects which are unrecognizable by healthy persons. Or they are excited to such a degree by certain odors, especially of flowers, as to give rise to hysterical attacks. These patients often manifest a penchant for disagreeable odors, such as that of asafœtida, burnt feathers, etc. There may also be diminution or abolition of olfaction on one or both sides, the former particularly in hemianæsthesia; this may be associated with anæsthesia of the nasal mucous membrane. Sneezing spasms are observed occasionally.

Similar symptoms are noticed on the part of the organ of taste. The patients are often capable of tasting the minutest trace of this or that substance; they acquire an unconquerable antipathy to certain articles, a predilection for others (sometimes for ill-tasting substances). They sometimes have a craving for indigestible articles, such as ink, chalk, lead pencils, etc. (pica hysterica).

Among the disturbances of individual organs, those of the digestive apparatus are probably the most frequent. These often begin in the buccal cavity. We have previously mentioned that disturbances of deglutition may result from hysterical paralysis of the muscles of the palate and œsophagus. These disturbances become more marked if the tongue is paretic or paralyzed. This also interferes with articulation.

In some cases, profuse salivation occurs, either during an hysterical attack or independently. In other cases, there is an abnormal diminution of the salivary secretion. The patients then complain of dryness

and pain in the mouth; fissures form not infrequently, particularly on the tongue, and the mucous membrane of the mouth is unusually red.

Globus hystericus is an almost pathognomonic sign of hysteria, and is attributed to ascending peristaltic spasms of the muscular coat of the œsophagus. The patients experience a sensation as if a round body were ascending from the stomach along the œsophagus. The starting-point of the globus is sometimes referred lower, in the pelvic region. The symptom may develop spontaneously, or is produced by mental excitement, or it is a precursor of an hysterical seizure. It may be produced at times by compression of hysterical pressure points.

Spasms of deglutition sometimes occur on attempting to eat or at the mere sight of food (hydrophobia hysterica).

Eructations are observed not infrequently. They occur spontaneously and last for days, even weeks, or they are produced by irritation of hysterical pressure points.

There is often a great accumulation of gas in the stomach and intestines. The origin of the large amounts of gas is attributed by some writers to exhalations from the vessels of the mucous membrane or to rapid decomposition of the gastro-intestinal contents. In our opinion, it is the result of the swallowing of air, which, on account of temporary incontinence of the pylorus as the result of disturbances of innervation, passes rapidly into the intestines. The application of faradism, and methodical massage of the abdomen, will generally expel the gas through the mouth and anus, almost as rapidly as it has accumulated.

Some patients suffer from obstinate vomiting. Soon after ingestion, the food is rejected almost unchanged, and hardly anything may be retained. It is often surprising how well this is tolerated by the patient.

Hæmatemesis sometimes occurs, and even very large amounts of blood may be vomited. This is evidently the result of vaso-motor disturbances of the gastric mucous membrane. In some cases, the blood is discharged through the rectum.

These symptoms often arouse the suspicion of gastric ulcer, and the diagnosis is not always easy. The differential diagnosis is rendered especially difficult by the presence of gastric pain, which in other cases is the sole hysterical gastric symptom. The intensity of the gastralgia is sometimes so great that the patients writhe in pain. There are also epigastric pains which are located in the abdominal walls, and are recognized by their superficial position.

Many patients also present vigorous epigastric pulsations, which are the result of local vaso-motor changes in the abdominal aorta.

The liver and spleen may be tender in places, and occasionally swollen.

In one of my cases, hysterical attacks could always be produced by pressure on the spleen; in another patient, paroxysmal attacks of hepatic pain had led to the diagnosis of gall-stones.

Intestinal pain is a more frequent symptom, and is either the result of intestinal distention with gas, or of spasm of the intestinal walls. Borborygmus is also a frequent symptom in hysteria. Digestion is disturbed in many patients; constipation is generally present. Hysterical seizures may be followed by watery diarrhœa—the result of vaso-motor and secretory disturbances.

Among the hysterical changes in the respiratory apparatus, we have

already mentioned paralysis of the vocal cords and its sequelæ. Spasm of the vocal cords may also occur.

In some patients, there is marked anæsthesia, in others hyperæsthesia of the laryngeal mucous membrane. This is generally associated with a similar condition of the pharyngeal mucous membrane. In the former event, the patient tolerates very well the introduction of the laryngoscope and the contact of the sound with the inside of the larynx. Laryngeal hyperæsthesia is accompanied by frequent coughing. True asthmatic attacks are sometimes produced by hysteria. Paralysis of the diaphragm, generally of one-half, is observed occasionally, and this may put the patient in danger of suffocation. Spasms of the diaphragm are more frequent and, if clonic in character, are manifested by hiccough. Hysterical singultus sometimes continues for days and weeks. Carré has called attention to the occurrence of hysterical pulmonary hemorrhage; it must probably be explained as the result of vaso-motor disturbances.

According to Empereur, less oxygen is consumed by hysterical individuals; furthermore, oxygen is stored up in the system, because the patients do not exhale as much oxygen in the expired carbonic acid as is inhaled with each inspiration. Counard mentions paroxysmal painful swelling of the breast, which is not alone tense, but may also be red and unusually warm; it contains small nodules. The swelling sometimes occurs during hysterical seizures or with the menses.

The circulatory apparatus presents central and peripheral disturbances of innervation. Attacks of palpitation or cardialgia are not infrequent; even Basedow's disease has been attributed to hysteria. The pulse is extremely variable as regards strength and fulness within a short period, and, since the heart's action may be unchanged, it is reasonable to assume that this is the result of peripheral changes in the arteries. There is sometimes temporary inequality of the pulse in corresponding arteries on the two sides of the body.

The urine sometimes presents striking changes. The patients may suddenly void large amounts of clear urine of low specific gravity (urina spastica). This is especially apt to occur after an hysterical seizure. In rare cases, there is oliguria or even anuria hysterica. For days and weeks, the patients pass very little or no urine. In its stead, there is profuse watery emesis, the vomited matters containing larger or smaller quantities of urea, although too small in amount to act as a complete substitute for the renal excretion. Hysterical polyuria and oliguria are probably the result of secretory disturbances.

The patients often complain of vesical tenesmus, and are compelled to micturate every few minutes. Others suffer from retention, so that the catheter must sometimes be resorted to for weeks and months.

With regard to diseases of the sexual apparatus, we must determine whether the changes present are the causes or the results of hysteria. This is not always easy. For example, menstrual disturbances not infrequently precede the outbreak of hysteria, while in other cases they follow it. The symptoms of hyperæsthesia are often present. Ovarialgia has been previously mentioned. Uterine neuralgia is also observed at times. Hyperæsthesia of the vagina is sometimes manifested by intolerable pruritus vaginæ or insatiable sexual desire. In some patients, the hyperæsthesia is so great that coitus is intensely painful, and vaginismus is produced. In other cases, there is anæsthesia of the vaginal mucous membrane, and no voluptuous sensations are produced during

coitus. Some hysterical patients complain of watery secretions from the genitalia, evidently as a result of vaso-motor and secretory disturbances. The course of hysteria varies greatly when conception occurs in hysterical individuals. In some, the symptoms disappear at once, and may even remain absent for a long time after delivery. In others, the symptoms increase, or at first increase and then diminish.

I have found in several instances that women who remained hysterical during pregnancy give birth to children who died after repeated eclamptic attacks. One woman, who suffered from several hystero-epileptic attacks during pregnancy, gave birth to a child which presented marked chorea, and died of eclampsia at the age of two months.

Disturbances of the general condition hardly ever remain absent in hysterical patients. They feel ill, are moody and depressed, and often complain of obstinate insomnia. There may be complete anorexia or insatiable boulimia, sometimes attacks of increased thirst. Certain writers apply the term hysterical fever to paroxysmal increase of temperature without a material basis.

The psychical condition suffers to a greater or less extent. It is characteristic of these patients that they exaggerate their sufferings and endeavor to attract the attention of the physician and others to their malady. In doing so they do not shrink from lying and simulation. Frogs, snails, insects are shown and are claimed to have been vomited, or evacuated from the anus or vagina. Others claim to have vomited fæces; still others, that they have eaten nothing for weeks, until they are caught at night secretly satisfying their hunger. Complaint is occasionally made of fever. The thermometer in the axilla does, indeed, show an increase in the bodily temperature, but this results from friction of the thermometer between the folds of the shirt. Our suspicions should be aroused if the temperature alone is elevated, while pulse and respiration are unchanged. Some patients increase the frequency of the pulse by voluntarily increasing the rapidity of the respiratory movements. If the patients think themselves neglected, they sometimes mutilate themselves, push needles under the skin, or swallow them, make preparations for suicide, but generally in such a manner that they may be prevented from carrying out their object.

Many patients are unable to control their emotions. Slight causes produce laughing or crying spasms, or sad events cause laughing spasms and vice versa.

Delirium, melancholia, mania, and other grave psychopathics sometimes develop.

The occurrence of somnambulistic, ecstatic, and similar conditions may also be referred to. Some patients remain for months in a somnolent condition. Attacks of profound syncope are frequent.

It is impossible for us to describe all the combinations in which the various symptoms of hysteria may appear. Remissions and exacerbations are of frequent occurrence, the latter not infrequently at the period of menstruation, or after mental excitement.

The disease always runs a chronic course, and generally lasts for life. While it is probably going too far to say that every woman carries within her the germs of hysteria, it is nevertheless true that very slight causes suffice to make a woman hysterical. The disease is rarely the cause of death. This is most apt to occur in hystero-epileptic attacks, and in spasm of the vocal cords, or paralysis of the circo-arytænoidei postici, and

in attempts at suicide, which may terminate fatally, although generally not meant in earnest.

IV. DIAGNOSIS.—The diagnosis is generally easy. It is true that very few symptoms are pathognomonic of hysteria, but the ensemble, the frequent changes, and the sudden appearance of entirely opposite conditions generally leave no room for doubt.

V. PROGNOSIS.—The prognosis as regards recovery is not favorable, but the patient's life is rarely endangered. The more numerous the symptoms, the more severe the hystero-epileptic attacks, and the more marked the mental affection, the more unfavorable is the case.

VI. TREATMENT.—Prophylactic measures possess no slight importance. They must be employed particularly in individuals who come of hysterical and nervous families. The children should be generously nourished, hardened bodily and mentally, they should avoid over-application in school, and be kept away from hysterical individuals.

If hysteria has developed, we must first endeavor to meet the causal indications, and these vary, as a matter of course, with the etiology.

Friedrich recently obtained good effects from cauterization of the clitoris. Extirpation of the ovaries has also been performed.

Against the disease itself more can be done by moral treatment than by drugs. The best results will be obtained by him who secures the confidence of the patient.

Asafœtida, valerian, musk, potassium bromide, arsenic, gold, silver, copper, etc., have been recommended, but we have never obtained any special benefit from these remedies. Electrical applications (central and peripheral galvanization and faradization, and electric baths) often fail us.

Great caution should be exercised in the administration of narcotics.

I have often derived benefit from the daily employment of protracted lukewarm baths (30° R., thirty to forty minutes' duration), especially if irritative symptoms predominated. Liebermeister recommends cool baths (15° R.) followed by walking, until the patient feels warm.

Individual hysterical symptoms must sometimes be treated with local remedies. In hysterical paralyses, the patients must be compelled to exercise the limbs vigorously every day. In paralysis of the legs, for example, the patient is placed on her feet, supported under the arms, and, if necessary, dragged around with force. Hysterical contractures are treated by passive stretching of the muscles, massage, and the faradic current. In hysterical anæsthesia, the faradic brush often produces rapid effects. Upon the outbreak of hysterical spasms, we may order cold baths and douches, or the electrical brush.

12. *Cerebral Neurasthenia.*

1. The causes and significance of neurasthenia have been discussed on page 147. In some cases, cerebral symptoms predominate (cerebral neurasthenia); the combination of cerebral and spinal symptoms is called general neurasthenia; those cases in which vaso-motor symptoms predominate are known as vaso-motor neurasthenia.

2. Cerebral neurasthenia is characterized by ready excitability and exhaustion of the brain. The patients are generally moody, complain of headache; some suffer from insomnia, others from excessive somnolence. They feel incapable of bodily, and particularly of mental work, are confused and forgetful. Some complain of dizziness, dream a good deal at night, speak in their sleep, and suffer from excessive pollutions. They are unusually sensitive to bright light and loud noises. Others complain of impaired vision, flashes of light before the eyes, and

scotoma. There is not infrequently impairment of hearing and subjective auditory sensations.

Some patients feel anxious and oppressed in the presence of many individuals; others only feel well and free in company. Many are tortured by the most various forms of terror. Walking across a large space, a visit to theatres and concerts, a railway ride, etc., produce a feeling of fear and dread of danger to life.

The patients not infrequently change color very quickly, complain of shooting heat and then of coldness, perhaps of paræsthesiæ. The symptoms may be confined to circumscribed nerve tracts, or may be unilateral (hemineurasthenia).

Neurasthenia is often manifested by functional disturbances of individual organs. Persons who are compelled to speak a good deal soon suffer from a feeling of exhaustion in the larynx, hoarseness, and a sensation of tickling. The patients often complain of dyspnœa, and attacks of palpitation of the heart are frequent. There is diminished thirst and appetite, disturbances of digestion, flatulence, and changes in the urinary excretion.

3. The diagnosis, prognosis, and treatment are the same as in spinal neurasthenia. Very rapid results are often, though not constantly, obtained by electrical applications. The methods recommended are: Galvanism longitudinally, transversely, or obliquely through the skull, or a large electrode to the head, the other to the feet, or central galvanization; also galvanization of the cervical sympathetic or the cervical cord, faradization of the head, or the application of the faradic brush to large surfaces of the skin. At first, a weak current should be employed; the sittings should not be too long or repeated too quickly.

PART V.

DISEASES OF THE SYMPATHETIC.

Little is known positively concerning diseases of the sympathetic. Reliance was formerly placed upon anatomical changes to prove the frequency of clinically demonstrable diseases of the sympathetic; but this mode of proof has been discredited since Lubimoff showed that changes in the sympathetic may be found in most corpses.

Diseases of the sympathetic may exist independently, or they may be secondary to diseases of the brain or spinal cord. The latter is readily explained by the fact that the sympathetic receives certain nerve tracts from the cerebro-spinal axis.

We have previously considered several affections of the sympathetic, viz.: Basedow's disease, stenocardia, Addison's disease. We will add the following:

1. *Hemicrania. Migraine.*

I. ETIOLOGY.—Unilateral headache is as frequent as it is annoying, particularly in the female sex. The disease often develops in childhood; at all events, a predisposition is manifested from the age of fifteen to twenty-five years. In a case reported by Bohn, hemicrania seems to have been congenital. Heredity can often be demonstrated. Either hemicrania or neuroses of various kinds are apt to occur in the patient's family. The higher classes seem to be affected more frequently than the laboring classes. There is no doubt, from our own experience, that mental strain is connected with the origin of the disease in some instances. At other times, it is the result of acquired nervousness, such as is particularly apt to develop after chlorosis, anæmia, vital losses of all kinds, protracted diseases, excesses. It is often associated with hysteria. It is observed occasionally after infectious diseases, especially gout and rheumatism. It has also been known to develop during preg-

nancy, and disappear after delivery. Oppenheim recently called attention to the fact that hemicrania sometimes precedes for a long time marked tabetic symptoms. Hack attributes certain cases to morbid swelling of the inferior turbinated bone, and obtained good effects from treating the latter with the galvano-cautery. In not a few cases the cause remains unknown.

The individual attacks of hemicrania may be the result of menstruation, bodily or mental strain, excitement, overloading of the stomach, constipation, vivid impressions upon the senses, etc.

II. SYMPTOMS.—The symptoms begin suddenly, or they may be preceded by prodromata. The latter last one or more hours, sometimes several days. They consist of a feeling of discomfort, dulness in the head, rush of blood to the head, dizziness, nausea, tinnitus aurium, etc.

In some cases, the patients are awakened from sleep by the violent headache, others feel tolerably well early in the morning, but the dreaded symptoms gradually grow stronger and stronger, generally last until night, and do not disappear until sleep occurs in the following night. An attack rarely lasts more than one day.

The pain is described as dull, boring, or crushing; as a rule, the patients do not describe the shooting, tearing pains of other neuralgias. It sometimes has a throbbing character, and increases with each pulsation. The pain is generally confined to one side of the head, usually the left. In some patients, its situation varies in different attacks (hemicrania alternans), or it is originally unilateral, and then extends gradually to the other side. Even if the pain is unilateral, it often extends beyond the median line, while in other cases it does not reach the latter.

The patients sometimes localize the pain chiefly in the forehead, the temples, or the vertex, more rarely in the occipital region. In many cases, the entire side of the head is uniformly sensitive. In some patients, the pain also affects the back of the neck, and complaint is made of stiffness in this region. Painful pressure points are absent, but a larger area at the apex of the parietal bone is often tender on pressure. The intensity of the pain generally increases on coughing, straining, or bending over. Many patients complain of pain in the orbit and of impaired mobility of the eye.

The painful half of the head is often extremely sensitive to gentle contact with the skin, while strong pressure is often borne without pain. Gentle pulling upon the hairs is often very painful. In patients who have suffered from hemicrania for years, the hair upon the affected side of the head is often scanty, prematurely gray, occasionally dry and ragged. It is also said that the hairs sometimes become erect during an attack.

Very many patients are overcome by the severity of the pain to such an extent that they are entirely incapable of mental or bodily work. They are extremely sensitive to bright light and loud noises; many complain of flashes of light before the eyes, visual hallucinations, scotoma, and even hemianopsia has been described. Others complain of impairment of hearing, tinnitus aurium, or other abnormal auditory sensations.

Many patients are pale and feel chilly; the pulse is somewhat accelerated; they suffer from frequent yawning and eructations, the tongue becomes coated, and vomiting occurs. In other cases meteorism develops, the patients suffer from frequent desire to go to stool, and after

the attack pass unusual quantities of a pale, watery urine, in which I discovered albumin on several occasions.

The duration and number of the attacks vary greatly. Months and years sometimes elapse before another attack appears, or women experience an attack before or during, rarely after, each menstrual period, or they recur in series, and are then followed by comparatively long, free intervals. The disease sometimes persists for life; in women it not infrequently ceases spontaneously at the menopause.

In the majority of cases, the symptoms described are the only ones observed. In other cases, vaso-motor and trophic changes also occur, and can hardly be explained except as the result of implication of the cervical sympathetic, or the vaso-motor centre. This is also indicated by the presence of pressure points in the neck, corresponding to the superior and middle ganglia of the cervical sympathethic, and over the lowermost cervical or upper dorsal vertebræ. Not all cases, however, are the result of disturbances of the sympathetic.

The sympathetic form of hemicrania (vaso-motor hemicrania) may be the result of spasmodic or paralytic conditions. The former is known as hemicrania sympathico-spastica, the latter as hemicrania sympathico-paralytica. Mixed forms also occur.

Hemicrania sympathico-spastica also presents symptoms, apart from the unilateral headache, which indicate tetanus of the vessels of the head supplied by the cervical sympathetic. The affected half of the face is unusually pale and cool. The temperature in the external auditory canal may be diminished 0.6° C. The temporal artery appears narrow and hard. The pupil on the painful side is dilated, the eye sunken. Pressure on the carotid of the affected side increases the pain, compression of the one on the healthy side generally diminishes it. Active salivation is sometimes observed; in one case Berger collected two pounds of saliva during a single attack. As the tetanus of the muscular fibres of the vessels subsides towards the close of the attack, the walls of the vessels relax, the face reddens and grows warm, the conjunctiva is injected, the secretion of tears increased, and the pupil narrowed. Many patients also complain of a general feeling of increased heat, of acceleration of the pulse, and palpitation of the heart; vesical tenesmus is experienced, and the patients pass large quantities of watery urine or sometimes thin stools. Among late sequelæ of the disease are mentioned obliteration of the folds of the skin upon the affected half of the head, thickening of the walls of the temporal artery, thickening and nodular formations of the skin.

The symptoms of hemicrania sympathico-paralytica remind us of those produced by section of the cervical sympathetic in animals. The painful side of the head is red and hot, the pupil is narrow, the eye sometimes sunken, and the palpebral fissure narrowed; there is sometimes slight ptosis. During the attack, Moellendorf observed a scarlet color of the fundus of the eye, redness of the papilla, the boundaries of which were somewhat effaced, abnormal fulness of the retinal arteries and veins, sinuosity and nodular distentions of the retinal veins, and injection of the episcleral vessels. Increased secretion of sweat (epidrosis unilateralis) is sometimes observed upon the affected half of the head and face. The temporal artery and, in many cases, the carotid are dilated and pulsate vigorously. Pressure upon the latter may relieve the pain, compression of the carotid on the healthy side increases the pain. Berger demonstrated increased tactile sensibility of the integument of

the affected side during the attack. The pulse is sometimes unusually slow, the radial artery small and hard. Towards the end of the attack, the redness of the face disappears, and is replaced by pallor and coolness, and dilatation of the pupil as the results of secondary narrowing of the vessels.

III. ANATOMICAL CHANGES AND PATHOGENESIS.—Nothing is known concerning the anatomical changes in hemicrania.

In the large proportion of cases, in which sympathetic symptoms are absent, the pathogenesis remains obscure. Much also remains unexplained concerning the sympathetic forms. Even the localization of the pain is unsettled—whether in the dura and pia mater or in certain provinces of the central nervous system. Du Bois-Reymond believes that the pain is the result of compression and irritation of the sensory nerves in the walls of the vessels, owing to the spasm of the vessels. Eulenberg attributes it to the numerous changes of intracranial pressure resulting from the changes in the amount of blood within the skull.

IV. DIAGNOSIS.—The diagnosis of hemicrania is easily made. It could only be mistaken for trigeminal or occipital neuralgia, but this is distinguished by the presence of painful pressure points. If distinct sympathetic disturbances are present, we can readily distinguish between the spastic and paralytic forms of the disease.

V. PROGNOSIS.—The prognosis is good so far as regards danger to life, but bad as regards recovery. As a rule, the disease cannot be relieved permanently. It generally disappears spontaneously with advancing years, and in women at the menopause.

VI. TREATMENT.—Prophylactic measures should be adopted in individuals who possess an hereditary tendency to the disease. In such individuals the mental and physical education should be such as to avoid, as much as possible, the causes of the disease.

Such measures should also be continued in order to prevent the recurrence of attacks. Light, nourishing diet, daily evacuation of the bowels, avoidance of mental and bodily overwork, constitute important elements of treatment. In anæmic individuals iron preparations, in nervous patients the nervines are indicated. Very good effects are often obtained from a change of air. Some patients feel better at the seashore, others in the mountains. In puffy, plethoric individuals, with a tendency to disturbances of digestion, cures at Kissingen, Homburg, Carlsbad, etc., are indicated.

Electricity is often resorted to, the galvanic current transversely and longitudinally through the skull, galvanization of the neck or cervical sympathetic, treatment of painful points with the anode. In the spastic form the anode, in the paralytic form the cathode should be applied to the cervical sympathetic. The faradic current has also been employed, particularly the primary current. Good effects have been obtained from the electrical hand, the patient taking one electrode in his hand, the physician grasping the other one and then stroking the head and face with his moistened free hand. Electrical baths and general faradization have also been recently recommended.

To relieve the individual attack, the patient should remain in a quiet, easily darkened room, and should be kept free from noise and excitement. He should assume the horizontal position, with the head as low as possible. Many feel great relief after taking a cup of strong coffee, others from swallowing small pieces of ice. While the pains are increased in many cases after eating, in others great relief is felt after a hearty meal. It is often said that the pain improves after eructation

and vomiting, so that many patients put the finger into the throat in order to produce emesis. The pain is occasionally alleviated by strong pressure on the head, the application of an ice-bag, compresses of cold water or vinegar, applications of ether, chloroform, or turpentine, and inhalation of ammonia. In the spastic form, good results have been obtained from inhalation of amyl nitrite (3 to 5 drops), in the paralytic form from the subcutaneous injection of ergotinum Bombellon (one-half syringeful diluted with an equal amount of water). Nitroglycerin has been recently recommended.

2. *Progressive Facial Hemiatrophy.*

(*Facial Trophoneurosis. Neurotic Facial Atrophy. Prosospodysmorphia.*)

I. ETIOLOGY.—This term is applied to atrophy of the panniculus adiposus and skin of one-half of the face, which gradually increases and gives rise to serious deformity. The facial bones and muscles, the palate and tongue may also take part in the atrophy. The disease is not frequent. Lewin recently succeeded in collecting the reports of only sixty-eight cases—forty-one in males, twenty-seven in females. The right and left halves of the face were affected with equal frequency. As a rule, the disease begins from the age of ten to sixteen years, rarely beyond the age of twenty-five years.

The influence of heredity is not proven, but several of the patients came of nervous families, or had manifested other nervous symptoms (trigeminal neuralgia, facial spasm, headache, epilepsy).

The disease sometimes followed traumatism, which either affected the face directly, acted centrally (injury to the skull) or upon the cervical sympathetic. It has also been observed after infectious diseases (measles, scarlatina, small-pox, typhoid fever, but particularly diphtheria).

The first symptoms sometimes developed during the puerperal period. In some cases no cause can be discovered.

II. SYMPTOMS.—The symptoms develop unexpectedly, or they may be preceded by prodromata for months and even years. The latter consist of neuralgiform pains, paræsthesiæ of various kinds, and even cerebral symptoms (vertigo, headache, spasms), etc. In Emminghaus' case, twitchings of the muscles of mastication were described.

The transition from the prodromal to the manifest symptoms is sometimes formed by changes in the hairs (beard, brows, scalp). They become more scanty, and sometimes fall out entirely; their color becomes lighter, often even of a silver gray. These abnormalities sometimes occur only in patches; in other cases they follow the facial atrophy.

As a rule, the atrophy begins with the formation of light patches upon the integument of the face. Their color is cicatricial white, yellowish, or brownish-yellow. The originally white patches often assume a yellow or brownish color in the further course of the disease. At first there is generally only one patch, but this is followed by others. The patches may coalesce with one another. In the beginning they sometimes develop only along certain nerve tracts.

The patches become more and more depressed. The panniculus adiposus beneath them atrophies and the skin grows thinner. At the same time the latter is adherent firmly to the underlying bones, and

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sometimes desquamates very actively. Not infrequently the face is traversed by a series of deep, cicatrix-like places. In other cases, the atrophy is more uniform; while the healthy side appears blooming, the diseased side looks shrunken and old (vide Fig. 129). The eye is generally shrunken (atrophy of the orbital cellulo-fatty tissue) and the palpebral fissure is sometimes dilated, sometimes narrowed. On the affected side the mouth is sometimes slightly open, on account of atrophy of the orbicularis oris and the traction of the atrophic integument. The external nasal opening and the auditory canal are not infrequently somewhat enlarged. In many cases the face is drawn towards the diseased side.

Cutaneous sensibility is unchanged in the majority of cases. Some patients state that, in testing with a needle, a sensation is felt as if the

FIG. 129.

The face in left-sided progressive facial atrophy.

skin were coated with varnish or rubber. In a few cases, diminution of tactile sensation and of electro-cutaneous sensibility has been observed. Some patients complain of paræsthesiæ of the atrophic side of the face. The cutaneous temperature is unchanged. The secretion of sweat is unaffected at first, but in very advanced cases it is sometimes diminished or abolished. The production of sebum is very soon diminished or abolished. Laude noticed, in one case, diminution of the secretion of cerumen. The power of blushing is unaffected, and the arteries on the diseased side are normal. In one case, Eulenburg and Landois noticed a greater elevation of recoil in the carotid pulse curve on the affected side.

The atrophy sometimes attacks the bones, cartilages, and muscles. Virchow states that atrophy of the bones is especially apt to occur when the disease begins in early youth. The bones contain more or less deep

furrows or abnormal ridges, and are thinned and shortened. The maxillæ and hard palate are attacked with special intensity. Absence of a canine tooth or one or more molar teeth from the deformed jaws has been noticed in several cases. The nasal and auricular cartilages, sometimes those of the eyelids, may also undergo atrophy and deformity.

The tongue upon the affected side of the face is sometimes diminished in size and, when protruded, deviates to the diseased side. The muscular tissue of the soft palate may also undergo atrophy. In a few cases, the patients suffered from diphtheria, and the atrophic side was then affected to a more marked degree.

The facial muscles may also be attacked by atrophy. The electrical excitability of the muscles is unchanged. Fibrillary muscular twitchings were observed in a few cases.

The muscular atrophy generally remains confined to the muscles of the face, tongue, and palate. In one case, disturbances of articulation were observed, especially in the articulation of the letter *r*.

The organs of special sense remain intact. In one case, there was diminution in the power of hearing, and Wolff observed narrowing of the field of vision.

Brunner and Seeligmueller described irritative symptoms on the part of the cervical sympathetic: dilated pupils, pale skin, lowered temperature, absence of perspiration, and pain on pressure over the sympathetic ganglia.

In two cases, similar cutaneous changes appeared on the corresponding limbs. Two cases of bilateral facial atrophy have been reported.

The course of the disease generally extends over several years. Remissions sometimes occur; in other cases the disease comes to a standstill, but the deformity persists for life. There is no danger to life, but in one case Delamare observed increasing excitement, and finally insanity.

III. NATURE OF THE DISEASE.—No post-mortem examinations have been made. Bitot and Laude regard the disease as a local atrophy of the panniculus adiposus of unknown origin. But the fact that not alone the skin, but also the bones, cartilages, and muscles take part in the atrophy indicates that the causes are more general in character, probably vaso-motor or trophic. In our opinion, the disease is probably the result of trophic disturbances, which either originate directly from the cervical sympathetic or from disease of the trophic fibres in the trigeminus (particularly in the spheno-palatine and Gasserian ganglia) and the facial nerve.

IV. DIAGNOSIS.—The diagnosis is easy. The disease is distinguished from congenital asymmetry of the face by its later development, the presence of pigmented patches and changes in the hairs. These features will also differentiate the disease from the acquired facial atrophy of scoliosis and caput obstipum.

V. PROGNOSIS.—The prognosis is unfavorable as regards recovery, since treatment has been entirely unavailing. Peripheral and central electrization have been employed without benefit.

3. *Facial Hemihypertrophy.*

1. Unilateral facial hypertrophy is extremely rare. Lewin recently collected ten cases.

The disease is always congenital. The progress of the disease after birth kept pace with the general conditions of development.

2. The hypertrophy affected particularly the soft parts of the face (vide Fig. 130), and also the ear, tonsil, and tongue. Hypertrophy of the teeth, upper and lower jaws was observed in one case. The sebaceous secretion was increased, and the follicles projected as little nodules, or the sebum had collected in places in the shape of crusts and scales. There was generally a considerable flow of saliva, in one case active secretion from the otherwise unchanged ear. The cheek was sometimes reddened and felt warm subjectively. Blood vessels, organs of special sense, and secretion of sweat unchanged. In Friedreich's case, there was diminished sense of taste on the hypertrophic half of the tongue. Other noticeable features were pigmentation of the skin, increased growth and abnormally dark color of the hair, occasionally diminished power of mimicry. Mental functions generally intact; in one case hydrocephalus and general convulsions.

An autopsy was obtained in Friedreich's case, but the results were negative.

In some cases the hypertrophy also involved the corresponding half of the

Fig. 130.

Left-sided facial hypertrophy in a girl æt. 9 years. After Schieck.

body. In one case the left side of the face, left arm, and right leg were hypertrophied.

4. *Irritative Conditions of the Cervical Sympathetic.*

I. SYMPTOMS.—Among the symptoms of irritative conditions of the cervical sympathetic, dilatation of the pupil or mydriasis spastica (the result of contracture of the dilator pupillæ) is the most frequent. Enlargement of the palpebral fissure and the protrusion of the eyeball are sometimes noticed on the affected side (irritation of Mueller's muscle). The power of accommodation suffers not infrequently.

Vaso-motor changes are often overlooked on account of their fleeting character. They are manifested by pallor of the corresponding half of the face, diminution of temperature (0.9° C. in the external auditory canal), and diminution or abolition of perspiration. The temporal and carotid arteries are sometimes less full on the affected side.

Trophic changes sometimes appear very early, and in one case had advanced so rapidly within a week that the emaciation of the cheek was noticed by the patient. It is uncertain whether these changes must be attributed to special trophic fibres or to the diminished amount of blood in the arteries.

The cervical sympathetic and its ganglia are sometimes tender on pressure.

The symptoms may be temporary or persist for life. Czermak and

Gerhardt observed a case in which oculo-pupillary symptoms alone occurred as the result of pressure on a tumor of the neck. Widd observed exacerbations and remissions during the course of a phlegmon of the neck.

Irritative and paralytic symptoms sometimes alternate with one another.

The patients feel very little discomfort, but may be disfigured by the extensive atrophy of the face.

II. ETIOLOGY.—Among the chief causes are diseases of the organs of the neck, associated with pressure upon or extension of inflammation to the cervical sympathetic (glandular tumors, phlegmon of the cellular tissue, parotid tumors, goître, aneurisms, fall, blow, stab or gunshot wounds in the neck).

The symptoms sometimes occur in diseases of the cervical cord, from which the oculo-pupillary sympathetic fibres take their origin (fracture, dislocation, exostoses, and tumors of the vertebræ, inflammation, softening, and hemorrhage of the cervical cord).

Perhaps the disease occurs occasionally as an independent neurosis.

III. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is not difficult. If goître and palpitation of the heart are also present, protrusion of the eyeball might lead us to mistake the condition for Basedow's disease; but in the latter the symptoms are almost always bilateral.

The prognosis and the treatment depend upon the primary disease.

5. *Paralytic Conditions of the Cervical Sympathetic.*

I. SYMPTOMS.—Oculo-pupillary, vaso-motor, and trophic symptoms also occur in paralytic conditions of the cervical sympathetic; the first are the most constant.

The pupil on the affected side is narrow (paralytic myosis), and it is sometimes oval instead of round. It reacts to light, but often slowly. The pupil dilates under the action of atropine, but not to such a marked extent as the other one; if both pupils are treated with calabar, the affected one contracts more markedly than the healthy one.

Slight ptosis is sometimes noticeable. We also find narrowing of the palpebral fissure and sinking in of the eyeball; the latter is often especially marked in the later stages of the disease, and is then the result of atrophy of the orbital cellular tissue. Diminution of intraocular pressure and secondary flattening of the cornea are sometimes observed. Myopia may be produced by disturbances of accommodation following paralysis of the muscles of the iris.

Vaso-motor changes are shown by increased fulness and sinuosity of the vessels, redness, increased warmth, subjective feeling of heat, increased production of sweat, tears, and saliva on the affected side. Elevation of temperature cannot always be demonstrated objectively. The symptoms mentioned sometimes extend to the cervical and upper thoracic region. Horner and Nicati distinguish two stages of paralysis of the sympathetic; in the second, the vaso-motor symptoms are said to become converted into the opposite ones: sensation of diminished heat, slighter fulness of the arteries, pallor, and anhidrosis.

To the latter stage belong the trophic changes which are characterized by emaciation of one side of the face.

Central symptoms are sometimes observed, and are attributed to in-

creased blood supply to one cerebral hemisphere. These include unilateral or bilateral headache, dizziness, and even impairment of memory.

Subjective symptoms are absent in the majority of cases, unless produced by the primary disease.

II. ETIOLOGY.—The causes are similar to those of irritative conditions of the cervical sympathetic: injuries (fall, blow, etc.), tumors of the lymphatic glands, and the parotid, phlegmons in the neck, goître, etc. It is sometimes observed in phthisis as the result of adhesions between the sympathetic and the apex of the diseased lung. It also occurs in diseases of the cervical spine and spinal cord.

Attention has been called to the frequent association of paralysis of the cervical sympathetic, and of the brachial plexus, as the result of coincident injury to the cervical sympathetic, or to the communicating branches between the sympathetic and brachial plexus. It is probable that the disease may also develop as an independent rheumatic affection.

III. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is easy. Prognosis and treatment depend on the primary disease. Otto effected a cure by galvanization of the cervical sympathetic, but recovery is generally unattainable.

To reach the superior cervical ganglion with the electrode, the latter should be passed in between the angle of the jaw and the outer end of the greater cornu of the hyoid bone. It is advisable to place the cathode in this region, while the anode is applied to an indifferent spot, or upon the other side of the cervical spine at the level of the fifth and seventh vertebræ.

6. Symmetrical Gangrene.

I. ETIOLOGY.—The disease has been observed most frequently in women; a certain predisposition is created by anæmia, delicate constitution, and nervousness. Some patients mention a cold as the cause of the disease. Nedopil reports a case in which this cause was associated with mental excitement. The affection has also been observed after infectious diseases (typhus, intermittent fever, syphilis). In some cases the disease itself creates the impression of an infectious process.

II. SYMPTOMS.—The symptoms appear most frequently in the toes and fingers, more rarely in the nose and ears. In a few cases the symptoms have also been observed upon the integument of the chest.

A feeling of coldness, then a livid, cyanotic color, paræsthesiæ, and blunted sensibility, more rarely hyperæsthesia, sometimes neuralgiform pains open the scene. At first the symptoms present remissions and exacerbations, later they increase in intensity. Subcutaneous ecchymoses or vesicles develop in some cases. In such places the skin then becomes blackish and undergoes gangrene, which may lead to the loss of the phalanges, or lay bare large parts of the surface. The symmetrical distribution of the gangrene on both sides of the body is especially noteworthy. The mucous membranes escape, but in one case I observed swelling and hemorrhages of the gums. High fever (41° C.) and enlargement of the spleen have been observed; Hameau found sugar in the urine. The symptoms may run an acute (one to two weeks), subacute, or chronic course.

The disease is regarded as a vascular spasm with secondary local asphyxia. Its symmetrical occurrence indicates that the spasm is located in the vaso-motor centre (medulla oblongata?).

III. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is easy. It is distinguished from the gangrenous form of ergotin poisoning by the previous history. The prognosis is serious, although the disease rarely proves fatal. Treatment: electricity (peripheral and central), massage, quinine, iron, if necessary surgical interference.

7. *Myxœdema.*

(*Cachexic pachydermique.*)

ETIOLOGY.—Myxœdema occurs chiefly in women (twenty-seven females in thirty-one cases collected by Morvan). As a rule, it does not develop before the period of puberty; it is rare in childhood and beyond the age of fifty years. It has been attributed most frequently to colds, also pregnancy, parturition, lactation and sexual disturbances in general, to atrophy of the thymus gland, and in one case to excitement. It is said to occur often in nervous individuals.

II. SYMPTOMS.—The symptoms consist, in the main, of cachexia, œdema, and nervous disturbances.

The most striking feature is the œdematous swelling which attacks the forehead, cheeks, eyelids, nose, and lips. The lids can only be half closed, and form thick sacs; the physiognomy changes so that the expression becomes stolid, animal, and coarse. Increased secretion of tears and saliva has been described in several cases.

The limbs also undergo considerable swelling and increase in size, the fingers and toes being most affected. The trunk may also present œdematous swellings. Unlike ordinary œdema, the pressure of the fingers leaves no depression in the skin, because the fluid accumulated in the cutis and subcutaneous cellular tissue contains a large amount of mucin, and has a semisolid consistence. The skin has an alabaster or waxy yellow color and feels cool. The patients also complain of a sensation of coolness, and even the bodily temperature has been found lowered in several cases (to 36.4° C.). The pulse is sometimes slow. The secretion of perspiration and sebum is often diminished, the skin dry and wrinkled; a few red patches are sometimes found.

The mucous membranes (mouth, larynx, digestive tract) are often thickened. The voice becomes hoarse, monotonous, sometimes indistinct and nasal.

Paræsthesiæ are frequent, and the changes in the integument may be preceded by pallor or a livid color. As a rule, the patients complain of anorexia and constipation; cachexia and albuminuria gradually develop. Then follow apathy, somnolence, muscular weakness, delirium, hallucinations, and mental impairment.

The disease runs a progressive, but chronic course (average duration, sixteen and one-half years).

Nothing is known positively concerning the nature of the disease. The view is gaining ground that it is nervous in its origin, some locating it in the vasomotor centre of the medulla oblongata, others in the sympathetic.

III. TREATMENT.—Massage, pilocarpine, peripheral and central electrization, quinine, iron, nourishing diet.

8. *Hydrops articulorum intermittens.*

I. SYMPTOMS.—This condition is manifested by paroxysmal swelling of the joints, especially of the knees. The swelling occurs at such regular intervals as to arouse the suspicion of malaria. The interparoxysmal periods vary from one to three or four weeks. The attack lasts four to six, sometimes even eight days. The swollen joint is free from inflammatory changes; in exceptional cases it is painful. The duration of the disease varies from several months to twenty-five years.

II. ETIOLOGY.—The causes of the disease are unknown. In two cases it was preceded by intermittent fever. It has also been observed in association with Basedow's disease and vaso-motor angina pectoris.

III. TREATMENT.—Quinine, arsenic, and ergotin have been employed in treatment. Pierson obtained good results from galvanization of the neck. Electrization of the crural and sciatic nerves and the joint itself should also be tried.

SECTION VI.

DISEASES OF THE MUSCLES.

1. *Pseudo-hypertrophy of the Muscles.*

(*Atrophia musculorum lipomatosa. Dystrophia musculorum progressiva. Juvenile muscular atrophy. Hereditary muscular atrophy.*)

I. ETIOLOGY.—The disease develops most frequently in children. The deformity of the muscles and limbs is sometimes noticeable immediately after birth (rare), sometimes the changes develop gradually at the age of two years or later. The majority of cases begin before the fifteenth year.

The male sex is most frequently affected (among one hundred and twenty-five cases, one hundred and three boys, and twenty-two girls and women).

The greatest etiological influence is exercised by heredity, and this disease probably includes all cases which have been described as hereditary progressive muscular atrophy.

It often affects several children in one family, although no other relatives had been attacked. In other cases, maternal relatives had been affected, but the mother, as a rule, escaped. In two cases, I observed heredity on the paternal side. The rarity of the latter occurrence is owing to the fact that males, when affected, either die or become impotent before arriving at a marriageable age. In nine of my cases, four occurred in Jews.

In the hereditary cases, the male sex is chiefly affected.

The disease sometimes follows infectious diseases (variola, measles, scarlatina, diphtheria, typhoid fever). Some ascribe the disease to scrofula.

Injury, cold, and exposure are also mentioned as causes. Perhaps this explains the more frequent occurrence of the disease in the poorer classes.

In a number of cases, other nervous disturbances (idiocy, hydrocephalus, asymmetry of the skull, convulsions, etc.) were also present.

II. SYMPTOMS.—The chief symptom is increase of size, with diminished power, of certain groups of muscles.

If the disease is congenital, the deformity arising from increased volume of the muscles is occasionally noticeable immediately after birth. In other cases it develops gradually. The patients first complain of a slight tired feeling in walking, and a tendency to fall. Dragging pains develop on account of the over-strain of the muscles. If the disease be-

gins before the children are able to walk, this power is sometimes learned very late (fourth or fifth year).

The increased volume of the muscles gradually becomes more prominent. It is most marked in the calves, next in the extensors of the .highs and in the buttocks; then it may extend to other groups of muscles. The muscles of the lower limbs are often pseudo-hypertrophic, while those of the back and upper limbs are markedly atrophied (vide Fig. 131). In rare cases, the pseudo-hypertrophy extends to all the muscles, even to those of the face. Hypertrophy of the tongue has also been observed, and gives rise to difficulty in speech and deglutition.

The more the size of the muscles increases the more marked become the disturbances in their function. These are espe-

cially noticeable in walking and standing. In walking, the patients lift the foot unusually high, on account of the drooping of the toes. The gait becomes waddling, the axis of the pelvis and trunk being moved forcibly from one side to the other with every step. Atrophy of the muscles of the back generally produces marked lordosis in the lumbar spine, while there is well-marked kyphosis in the dorsal region. Weakness of the muscles of the buttocks and back interferes with sitting down and rising. In sitting down, the patients drop like an inert mass; in rising, they aid themselves by means of the hands and arms. If placed on the floor, they rise by climbing up their own body (placing the hands progressively higher and higher upon the thighs).

In dorsal decubitus, the foot is generally in pes varo-equinus position. The hip and knee joints are often flexed, the thigh abducted.

The muscular changes are generally, though not always, equally far advanced on both sides of the body.

The hypertrophic muscles are generally soft and flabby, more rarely they are firm and hard (marked proliferation of the interstitial connective tissue).

Fibrillary twitchings are absent. The mechanical excitability of the muscles is abolished in very advanced cases.

The electro-muscular excitability diminishes, and finally disappears with the increasing disappearance of the muscular structure.

Position of the body and appearance in pseudo-hypertrophy of the lower limbs, with atrophy of the back muscles. After Duchenne.

The skin over the affected muscles is often bluish-red and marbled, and is cold to the feel (even 9° C. lower than the axillary temperature). The production of heat in the diseased muscles is diminished. The congestion of the skin has been attributed to compression of the vessels in the muscles. Cutaneous sensibility is unchanged. The skin presents a great tendency to inflammation, so that slight pressure may suffice to produce gangrene and suppuration. The subcutaneous adipose tissue is almost always very abundantly developed. There is diminution of perspiration and active desquamation of the epidermis. The patellar tendon reflex was absent in some cases.

As a rule, appetite and sleep are normal, the bladder acts promptly.

Constipation is often present, especially if the abdominal muscles are paralyzed. The patients often suffer from bronchitis and may die from pulmonary complications (weakness of the respiratory muscles). Cardiac hypertrophy has been observed in a number of cases, and the pulse is sometimes slow (forty to sixty beats a minute).

The mental faculties are often unaffected. In other cases apathy, even imbecility and idiocy have been observed.

Traces of leucin, tyrosin, and sugar have been found in the urine; the amount of urea is sometimes diminished. Polyuria is mentioned in a few cases.

The disease runs a chronic course and may last more than twenty years. The patients gradually lose the power of moving about and are confined to the bed. Death is the result of intercurrent diseases, paralysis of the respiratory muscles or increasing marasmus.

FIG. 132.
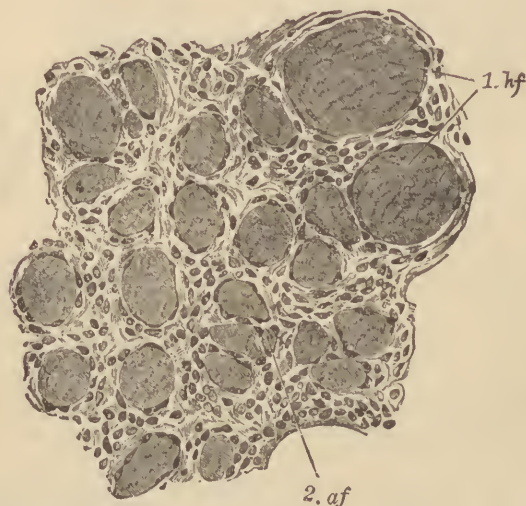
Beginning changes in lipomatous pseudo-hypertrophy of the muscles. After Ebstein and Marr. Increase and nuclear proliferation of the interstitial tissue and increase of the sarcolemma nuclei. 1. *hf*, two hypertrophic fibres ; 2. *af*, atrophic fibres. Enlarged 400 times.

IV. ANATOMICAL CHANGES.—The brain, spinal cord, and sympathetic have been found intact. In a few cases, lesions of the cord have been reported, but they were either accidental complications or unreliable. The peripheral nerves are generally unchanged, but a few writers have reported interstitial proliferation of fat (which also takes place under other circumstances), narrowing, flattening, and gray discoloration, proliferation of interstitial connective tissue, atrophy and disappearance of the nerve fibres. All these changes are probably the result of disuse of the muscles.

The muscles have a pale or butter-yellow appearance; in advanced cases they look like a lump of fat.

An abundant development of fat sometimes takes place in the fasciæ and tendons.

The majority of writers now believe that the process in the muscles begins with proliferation of the interstitial connective tissue. This starts from the in-

Fig. 133.
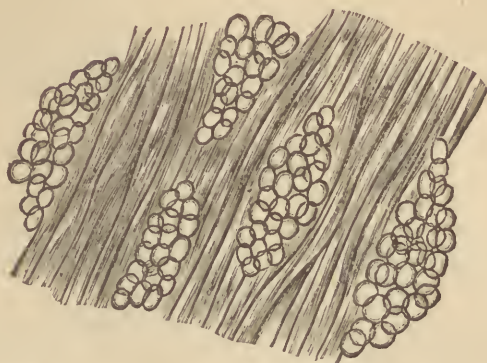
Advanced interstitial proliferation of fat. Slightly enlarged. After Leyden and Wernich.

ternal perimysium and the adventitious lymph sheaths of the blood-vessels. The individual muscular bundles are thus widely separated, and the interstitial connective tissue contains an unusual number of cells (connective-tissue and round

Fig. 134.

The same as Fig. 133, in transverse section. Enlarged 600 times.

cells). The more the interstitial tissue increases, the more the muscular substance disappears as the result of compression. In some cases, the process seems to stop at this stage, and the muscles remain very firm.

As a rule, adipose tissue develops in the new-formed connective tissue, the cells taking up fat and thus becoming converted into fat cells. The muscular fibres disappear more and more, and the muscle is thus converted finally into a clump of fat.

In the majority of cases, the fibres simply grow smaller until they disappear; sometimes they present the phenomena of Zenker's degeneration. Martini also described the formation of vacuolæ and canals, which were filled with albuminoid fluid. All these phenomena are also observed in other conditions.

In a few cases, the muscular fibres are in a condition of cloudy swelling, fatty degeneration, and nuclear proliferation. Cohnheim also described hypertrophic muscular fibres (singly and in groups), some of which had undergone dichotomous division.

The changes in the muscles are best followed in small pieces which have been removed from the living subject by the harpoon or knife.

Different views are entertained concerning the nature of the disease. In our opinion, it is myopathic in its origin. The theory of its identity with progressive muscular atrophy is combated by the following facts: *a*, Pseudohypertrophy occurs almost exclusively in youth; *b*, it always begins in the lower limbs; *c*, the electrical irritability of the muscles is different; *d*, it is exquisitely hereditary.

IV. DIAGNOSIS.—The recognition of the disease is easy except at the outset, when the examination of the muscle may be necessary to clear up the case.

It must be differentiated from: *a, spinal progressive muscular atrophy :* pseudo-hypertrophy does not begin in the interossei, the thenar and hypothenar eminences, but in the lower limbs; it is often hereditary, begins at the beginning of puberty, and does not present fibrillary contractions, or degeneration reaction; *b, acute spinal infantile paralysis,* with secondary hyperplasia of the panniculus adiposus; in the latter, the symptoms begin suddenly and are not progressive; the muscles do not react to the induced current; *c, spinal paralysis* and secondary development of fat in the paralyzed muscles; the paralysis *per se* exists for a longer period and to a more marked degree than is accounted for by the proliferation of fat.

V. PROGNOSIS.—The prognosis is unfavorable, the few reports of improvement or recovery being unreliable.

VI. TREATMENT.—The most promising measures appear to be massage, faradization, galvanization, and alcoholic inunctions of the affected muscles. Little can be hoped for from potassium iodide, arsenic, and other nervines.

2. *True Hypertrophy of the Muscles.*

I. ANATOMICAL CHANGES.—A few cases of this disease have been observed. The muscles increase in size from enlargement of the individual muscular fibres. Auerbach found the hypertrophic fibres of the biceps and deltoid muscles 165 μ and 120 μ in width, while fibres of corresponding muscles in other bodies were only 75 μ and 110 μ wide (1 μ = 0.001 mm.). The transverse striation is intact or unusually distinct. The muscle nuclei are enlarged and increased in number; no changes in the interstitial connective tissue.

II. SYMPTOMS.—The symptoms develop gradually. In Berger's case they were preceded by neuralgiform pains and paræsthesiæ; this case also presented objective sensory disturbances, which are generally absent. The limbs are usually affected, most frequently the muscles of the arms, next those of the lower limbs (particularly the calves and extensors of the thighs), next those of the trunk. The disease may be unilateral or bilateral. The muscles are unusually large, but their power is generally diminished. This is supposed to be owing to the fact that the blood-vessels no longer suffice to nourish the hypertrophic fibres, perhaps also the intact terminal plates of the nerves no longer prove sufficient. Auerbach noticed increased power during movements of brief duration, and Friedreich found the muscular power unchanged in a case in which some of the muscles

were atrophied. Berger described fibrillary contractions. The electrical excita-
bility is generally unchanged, though occasionally diminished. Benedikt noticed
increased mechanical excitability of the muscles. This writer also observed. as a
complication, vaso-motor disturbances (livid color, sensation of cold) and paralysis
of the sympathetic (redness of the face and unilateral hyperhidrosis).

III. ETIOLOGY.—The causes mentioned are typhoid fever, overstrain of the
muscles, and injuries. In Friedreich's case, the disease was congenital.

IV. DIAGNOSIS, PROGNOSIS, TREATMENT.—With the aid of microscopic exami-
nation of the excised piece of muscle, the diagnosis is easy. The prognosis is
grave, as the disease advances uninterruptedly. Treatment has been hitherto
useless.

3. *Progressive Ossification of the Muscles. Myositis ossificans progres-siva s. multiplex.*

I. ETIOLOGY.—Hardly more than twenty-five cases of this disease
have been reported. It generally begins during childhood (before the
age of fifteen years), once it appeared at the age of eight months; it
almost always begins within the second decennium. The male sex is
affected more frequently than the female. The causes are generally
unknown; the disease has been attributed to cold, injury, and strain.

II. SYMPTOMS.—The first changes generally appear in the muscles
of the nape of the neck and back. Then they extend to the muscles of
the neck, shoulders, and arms, later to the lower limbs. Even the mus-
cles of mastication, the facial and palatal muscles may be affected. The
abdominal muscles, tongue, laryngeal muscles, sphincters, muscles of the
genitals, diaphragm, heart, and small muscles of the hand are never
attacked.

The process may or may not begin simultaneously in the correspond-
ing muscles on both sides of the body. It runs a chronic course, and
may last more than twenty years. It is characterized by paroxysms, the
intervals varying from weeks to years. The attacks generally occur
without any known cause, more rarely they follow an injury.

Each attack begins with severe pain in a circumscribed part of the
muscle, but the pain not infrequently radiates over a large area. The
skin over the painful part is hot, red, tense, and œdematous, the œdema
often being very extensive. There is often more or less rise of tempera-
ture. These symptoms subside in a few days, and a tense, usually
doughy tumor can then be felt over the painful spot. In favorable cases
this disappears in a few days, leaving behind stiffness and impaired
mobility.

In other cases the swelling becomes hard, the muscle undergoes con-
tracture, and gives rise to deformity. The process corresponds anatom-
ically to the formation of a connective-tissue muscle callosity.

In still other cases, ossification ensues. A hard, at first movable body is
felt, which gradually increases in size. In this way the muscle is replaced by
bony tissue. The bony masses may be round, nodular, flat, or irregularly stellate.
They gradually adhere to the underlying bone, and become immovable. The
bones themselves often contain multiple exostoses.

If the muscles of the back are affected, the patients are stiff as a rod, are una-
ble to turn and lie down. Disease of the muscles of the neck and throat renders
the head immovable. Affection of the thoracic muscles acts like a firm cuirass,
produces dyspnœa, and permits breathing by the action of the diaphragm alone.
Ossification of the muscles of mastication interferes so seriously with the inges-
tion of food as to render artificial feeding necessary. Finally the patient may
become immovable, even passive movements being impossible.

Some points of ossification occasionally soften and disappear, even within
eight or ten days. In one case, Davy found diminution of the phosphate of lime

in the urine. Gerhardt and Peter observed normal amount of urea, diminution of uric acid, earthy phosphates, and kreatinin.

The patients generally die from inanition or from diseases of the respiratory apparatus and suffocation.

Tachycardia and unilateral hyperhidrosis of the head, tabes dorsalis and polyuria have been observed as complications.

III. ANATOMICAL CHANGES.—The process is supposed to run its course in the following manner; *a.* inflammatory proliferation of the interstitial connective tissue. *b.* Compression atrophy of the muscular fibres. *c.* Bone formation in the newformed connective tissue. The osseous substance is provided with periosteum and nutritive canals for the blood-vessels. The formation of cartilage is mentioned in one case.

IV. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is easy. The disease is distinguished from other bony tumors of the muscles by its progressive, multiple character. The prognosis is unfavorable.

Treatment: at first antiphlogosis, particularly rest, ice-bags, and potassium iodide internally. After relief of the acute symptoms, careful massage, iodine baths, inunctions with tincture of iodine, potassium iodide, iodoform, or mercuria ointment.

Mercury, sarsaparilla, guaiac, colchicum, nitric acid, lactic acid, and phosphate of lime have been administered internally.

4. *Ischæmic Muscular Paralysis.*

It is well known that paralysis and contracture develop not infrequently after the application of tight bandages, and have been attributed to ischæmia of the muscles. It has been shown that such paralyses may be produced in animals, and that they correspond microscopically to myositis. Poensgen recently reported a case in which a cold is said to have been the cause of purely myogenic paralysis.

SECTION VII.

DISEASES OF THE SKIN.

PART I.

INFLAMMATIONS OF THE SKIN.

DERMATITIDES.

a. ERYTHEMATOUS INFLAMMATIONS OF THE SKIN.

1. The chief manifest symptom of erythematous dermatitis is redness of the affected portions of the skin. This may also be the result of simple cutaneous hyperæmia, so that we distinguish erythema hyperæmicum simplex and erythema exsudativum. In the latter, the distention of the vessels is associated with exudation of a serous, sometimes of a bloody fluid in the immediate vicinity of the cutis. Hence, pressure upon the skin leaves behind a yellowish or hemorrhagic patch.

Simple erythema often is converted into the exudative form; the latter is sometimes an intermediate stage, which leads to the formation of vesicles and even of necrosis of the skin.

2. Erythema exsudativum is divided into three groups, according as it depends on local, toxic, or general causes.

Exudative erythema from local causes has the same etiology as hyperæmic erythema, viz., injuries, heat, the action of caustic substances.

Toxic exudative erythema is that form which generally follows the administration of medicinal agents, such as quinine, salicylic acid, antipyrine, chloral hydrate, strychnine, digitalis, copaiba, opium (medicinal exanthems). The redness of the skin is either diffuse or patchy, the patches being sometimes more or less elevated. Some persons possess a distinct idiosyncrasy with regard to such eruptions.

We will now discuss in detail the erythemata produced by general causes.

1. *Nettle Rash. Urticaria.*

(*Cnidosis.*)

I. SYMPTOMS.—Urticaria consists of the development of wheals (pomphi) on the skin. These consist of elevations whose surface is more extensive than their height, and which may attain the size of the palm of the hand. They are either red or pale in the centre and surrounded by

a red periphery. They itch, and disappear very rapidly without desquamation, in rare cases are followed by slight pigmentation.

The skin between the wheals may be normal or reddened. The wheals often may be produced by irritating the intact skin with the nail or other hard substance. If a wheal is pricked and compressed upon the the sides, a little drop of clear or bloody serum exudes, and the wheal grows distinctly smaller. This indicates that the process is the result of inflammatory transudation in the superficial layers of the cutis.

The wheals are sometimes so closely aggregated that they coalesce (urticaria conferta), or they are arranged in chain-like, but irregular rows (urticaria gyrata). In rare cases, the wheals develop into firm nodes (urticaria tuberosa) or form small itchy papules (urticaria papulosa s. lichen urticatus). Finally, the epidermis over the wheals may be raised like a vesicle (urticaria miliaris).

The wheals are most abundant in the face and trunk, next on the neck and limbs. On the lids, lips, and prepuce they are often associated with distinct œdema of the skin. They may appear at night and disappear during the day.

Wheals may also form on the mucous membrane of the cheeks, uvula, epiglottis, and œsophagus. Trousseau's experience that cutaneous urticaria may alternate with asthma-like attacks arouses the suspicion that the lower air-passages may also be attacked.

The eruption is preceded not infrequently by febrile, gastric prodromata. The patients feel chilly, the bodily temperature is increased, there is sometimes a peculiar feeling of anxiety and shortness of breath. In addition, they suffer from anorexia, coated tongue, diarrhœa, or obstinate constipation. At the end of a few hours or days, the characteristic eruption appears. In some cases, the general symptoms appear after the outbreak of the eruption; in others, they are rudimentary or entirely absent.

The subjective symptoms consist mainly of intolerable pruritus, which is increased especially by warmth. In two cases, Leube observed albuminuria during the eruption.

The individual wheals generally last a very short time, often only a few minutes, but others are constantly sprouting up on other parts of the integument. The entire eruption may last only a few hours, or it continues for several days, or relapses occur at certain intervals, or finally the disease runs an uninterrupted chronic course for several years.

II. ANATOMICAL CHANGES.—In wheals produced in rabbits, J. Neumann found œdema of the superficial layers of the cutis, swelling of the lowermost cell layers of the rete Malpighii, and anæmia of the cutis vessels. In wheals removed from the human subject, Vidal found dilatation of the blood-vessels and lymphatics, and white blood-globules outside of the walls of the vessels, partly in groups in the meshes of the connective tissue, partly between the cells of the rete Malpighii.

III. ETIOLOGY.—The causes of the disease may be external (local) or internal.

The former include cutaneous irritants, such as contact with the stinging nettle, rhus toxicodendron, certain caterpillars and molluscs, the stings of insects (fleas, bedbugs, flies, etc.), application of Lister's gauze, exposure to a cold wind, etc.

Many individuals possess such sensitive cutaneous nerves that, although they do not suffer from urticaria, any desired figure upon the skin may be produced

by contact with a hard substance (urticaria factitia). The irritated spot first be-comes pale, then red, and finally a wheal develops.

Among the internal causes is the ingestion of certain drugs and arti-cles of diet. The mere contact of these substances with the buccal mu-cous membrane is sometimes sufficient to produce the urticaria. This is evidently the result of reflex nervous influences. The substances in ques-tion include strawberries, fat meats and sausages, certain kinds of wine, oysters, lobsters, mussels, herring, vinegar, etc., or quinine, antipyrine, opium, chloral hydrate, arnica, cubebs, copaiba, turpentine, etc.

In other cases, the reflex irritation starts from the gastro-intestinal mucous membrane (gastro-intestinal catarrh, worms).

Urticaria occurs not infrequently in diseases of the uterus and ova-ries. It may appear at the menstrual period, or even take the place of menstruation.

It is sometimes observed in general diseases (cancer, phthisis, dia-betes, Bright's disease) and in infectious diseases (relapsing fever, typhoid fever, measles, scarlatina, intermittent fever).

Urticaria is not infrequent in jaundice, as the result of local cutane-ous irritants or of the violent itching and scratching of the skin. It has been observed in several cases of puncture of hepatic echinococci and in hepatic colic.

It may also be a prodrome of other skin diseases, such as prurigo and pemphigus.

Urticaria is sometimes produced by direct nervous influences, for example in individuals who have suffered from neuralgia, after violent emotions, and also in hysteria.

The disease sometimes appears epidemically in connection with epidemics of erysipelas. In not a few cases, no cause can be discovered.

Most authors are agreed that urticaria is an angioneurosis of the skin, but it must not be forgotten that much is left unexplained by this term.

IV. DIAGNOSIS.—The disease can hardly be mistaken for any other, but we must not remain satisfied until the special cause of the attack has been discovered.

V. PROGNOSIS.—Many cases recover in a short time. But it must not be forgotten that the chronic form of the disease may be severe, often incurable, and may drive the patient to distraction.

VI. TREATMENT.—The treatment must first be directed towards the removal of the exciting cause.

Not much can be expected from internal remedies; potassium bro-mide, arsenic, ergotin, and atropine have been recommended. Carbolic acid in pill form acted well in some of our cases. In intermittent urti-caria, quinine should be administered. Shoemaker cured a case of chronic urticaria with sulphuric acid.

Carbolic acid is the best external application; in a violent case, cool compresses of a five-per-cent solution; in milder cases inunctions of car-bol-vaseline (℥ ij.: ℥ xx.) or chloral hydrate ointment (℥ i. : ℥ xx.), t. i. d. We may also apply ether, chloroform, eau de cologne, lemon juice, or acetic acid.

The diet should be light, the clothing and bed covering not too warm.

2. *Erythema Nodosum.*

I. Symptoms.—In this disease, red, bluish-red or greenish nodes appear first, and in many cases exclusively, upon the anterior surface of the legs, their size varying from that of a hazelnut to that of a fist. The nodes are isolated, and the skin over them is destitute of folds, shining, and tense. They are warm to the feel and tender on pressure, and the surrounding integument is slightly œdematous. Similar ones occur not infrequently upon other parts of the limbs, more rarely on the face, or even the buccal mucous membrane.

Two cases are reported in which the nodes are said to have developed in the mucous membrane of the air passages.

The development of the efflorescence is sometimes remarkably rapid. It begins either as hyperæmic patches, or as red, wheal-like elevations, which gradually increase in extent and height. Later they assume a more hemorrhagic appearance, become bluish-red or green, then yellowish, and finally fade away. In rare cases, slight desquamation is left over.

In exceptional cases, the nodes undergo ulcerative degeneration. In others, the inflammatory process results in the production of vesicles and pustules. Purpura and wheals are sometimes found in addition to the characteristic nodes.

The general condition is sometimes very little affected, and the patient merely experiences a slight burning and pricking in the affected parts.

The outbreak of the nodes is sometimes preceded by anorexia, malaise, and febrile movement. The fever increases upon their appearance and the patients often fall into a depressed and tearful mood.

Pains and slight swelling of the joints are observed very often, and many believe that erythema nodosum and articular rheumatism bear intimate relations to one another. The articular affection is said to give rise, at times, to ankylosis. Endocardial murmurs are heard not infrequently; they are either anæmic or febrile, very rarely organic in character. The disease lasts three to four weeks, and almost always ends in recovery. In a few cases, fatal pleuro-pneumonia, inflammations of the serous membranes, and ulcerations of the mucous membranes have been observed as complications. In one case it was followed by miliary tuberculosis of the meninges. According to Oehme and Uffelmann, it offers a certain predisposition to pulmonary phthisis; according to others, it may be followed by endocarditis.

II. Etiology.—The disease occurs most frequently in young women, and is not rare in childhood. The patients are often chlorotic and feeble, or suffer from uterine diseases and menstrual disturbances; it sometimes takes the place of missed menses. Several cases have been observed after follicular angina. It occurs often in scrofula, and also in individuals who come of phthisical families. It may also be associated with syphilis, and Suessmann observed the disease run its course under the clinical history of quotidian intermittent fever. Recently I observed two cases after acute gonorrhœa. A certain predisposition to the disease is created by exhausting influences, such as protracted diseases. Erythema nodosum sometimes appears in the form of an epidemic, particularly in the autumn and spring, and in such cases is often associated with herpes and erysipelas.

We regard the disease as an infectious process, in which the cutaneous changes merely constitute one of the most striking symptoms. Its epidemic appearance and cyclical course, the severe constitutional symptoms, the intimate relations to affection of the joints and perhaps to endocarditis, the simultaneous occurrence of erysipelas and herpes, favor this view. Hebra regards the disease as an inflammation of the cutaneous lymphatics. Bohn explains it as the result of embolism of cutaneous vessels, and as identical with peliosis rheumatica. According to Purdon, it is the result of vaso-motor disturbances.

III. DIAGNOSIS.—The disease may possibly be mistaken for traumatism, small-pox, scarlatina, syphilis.

The occurrence of nodes on symmetrical parts, the swelling of the joints, and febrile general disturbance will serve to exclude trauma.

The differentiation from small-pox may be difficult. It is important to know whether variola is prevailing at the time. But erythema nodosum is not often attended with the formation of vesicles and pustules.

The disease cannot be mistaken for scarlatina unless carelessly examined, especially as the cutaneous lesions of erythema nodosum are not diffuse.

Syphilis is recognized by the previous history, the presence of syphilitic cicatrices and the results of treatment with mercury and potassium iodide.

IV. PROGNOSIS.—The prognosis is almost always good, except that the development of nodes upon the respiratory mucous membrane may prove dangerous, the joint affection may lead to ankylosis, and, according to some writers, valvular lesions may be left over.

V. TREATMENT.—The treatment consists of rest and nourishing diet; quinine in case of fever. Struempel obtained good results with salicylic acid. Local treatment is unnecessary, unless there is severe burning or pain in the nodes. We may then apply compresses of lead water, liq. ammon. acetici (5%), carbolic acid (2%), or may brush the nodes with collodion or iodoform ointment (ℨ i. : ℨ xx.).

3. *Multiform Exudative Erythema.*

(*Erythema Polymorphon.   Erythanthema Essentiale.   Herpes Iris.*)

I. SYMPTOMS.—Multiform exudative erythema is closely allied to erythema nodosum, but is more variable in its external appearances.

At first we find red patches, as large as a pea, which grow pale on pressure; they gradually increase on size and become raised. They almost always occur first on the dorsum of the hand and foot, then on the legs and forearms, then on other parts. The mucous membrane of the mouth, pharynx, and genitals may also contain raised hyperæmic patches, which may give rise to excoriations. In rare cases, the disease begins in the face and extends downwards.

The larger patches and nodes soon become cyanotic and red, and coalesce in part. They present transitions between blue, green, and yellow.

After a while the patches often undergo further changes. While the centre grows pale, the periphery remains injected (erythema annulare), new, injected patches sprout up at the periphery, or finally adjacent efflorescences coalesce and form twisted, garland-like figures. If the erythema retains its nodular form, it is known as erythema papulatum s. tuberculatum.

In addition, we sometimes find wheals, purpura, vesicles, and pustules. The nodes at the periphery are sometimes surrounded by a ring of vesicles (herpes iris).

In many cases, the disease runs a typical course, and is often preceded by the prodromata described in the consideration of erythema nodosum. A rise of temperature (even above 40° C.) sometimes continues for a certain length of time after the development of the cutaneous changes. Articular changes may occur, and likewise anæmic, febrile, rarely organic, cardiac murmurs. Enlargement of the liver and spleen has been observed, sometimes complications with pleurisy, pneumonia, albuminuria, and renal hemorrhage. The subjective symptoms are generally trifling: slight burning and pricking in the nodes.

As a rule, the disease lasts from one to two months; Lippe reports a case of eight months' duration. A fatal termination is exceptional.

II. ETIOLOGY.—The disease occurs mainly in young women who suffer from chlorosis, menstrual disturbances, or uterine diseases. A predisposition is also created by syphilis or cachexia. Lewin observed the disease frequently in women suffering from urethritis.

In our opinion, this disease, like erythema nodosum, is infectious. Like the latter, it occurs in epidemics in the spring and autumn, but in some individuals the disease returns at certain seasons of the year.

IV. DIAGNOSIS AND TREATMENT.—The same mistakes are possible that have been mentioned in describing erythema nodosum. The treatment is also similar to that of the latter disease.

4. *Acrodynia.*

(*Erythema Epidemicum.*)

In 1823 to 1830 this disease was observed as an epidemic in France, and it is also said to occur in the Orient.

It often began with gastro-enteritic symptoms: nausea, vomiting, anorexia, colic, and diarrhœa. An erythematous inflammation then appeared, first upon the hands, later extending along the limbs to the trunk. The formation of wheals and vesicles has also been noticed. The skin then desquamated and left behind a deep dark color, particularly over the chest and abdomen. In addition, disturbances on the part of the nervous system: a feeling of formication, tremor, paralysis of the limbs and bladder. Ophthalmia was a frequent complication. Duration of the disease, several weeks or months; fatal termination not infrequent. It is probably the result of poisoning with spoiled grain.

5. *Pellagra.*

I. ETIOLOGY.—The disease is endemic in certain parts of Spain, France, Roumania, but particularly in Northern Italy. In Lombardy, for example, more than thirty per cent of the population have been attacked. In 1879, nearly 98,000 individuals were attacked in Italy, nearly 41,000 in Lombardy alone. The disease also occurs sporadically in other localities.

Among the Italians the disease occurs only among the rural population, and almost always among those who use maize as an article of diet. Some attribute the disease to the non-nitrogenous character of maize, but it must be remembered that pellagra is sometimes absent in certain districts, despite the almost exclusive use of maize as an article of food. Others state that in the affected districts, the maize employed is unripe and mouldy. Attention has been called to the fact that the disease does not prevail in those countries in which maize is used only when dried. Opinions also differ as to whether the disease is produced by certain fungi, or by noxious chemical substances.

According to these views, the disease belongs to the same category as those produced by the ingestion of spoiled cereals. But it is said that pellagra may develop in those who never come in contact with maize, so that it is looked upon by some as the result of poor nourishment and bad surroundings. It is also claimed that the disease is hereditary.

II. Symptoms.—The chief symptoms are cutaneous changes, disturbances of digestion, and nervous disturbances. The disease is generally chronic and may last ten to fifteen years.

The first symptoms begin in April to June, and subside in autumn, to reappear more severely in the following year.

On those parts which are exposed to the sun, erythematous patches develop; these desquamate at a later period, and leave behind a dark olive discoloration. The skin becomes fissured and covered with ulcers, crusts, and nodes. The mucous membrane of the mouth and the nails become diseased. The patients suffer from fever and diarrhœa, complain of formication in the hands, muscular tremor, paralysis. Ptosis, hemeralopia, and diplopia make their appearance. The patients are profoundly depressed and insanity may develop. Death occurs finally from increasing exhaustion.

III. Anatomical Changes.—These are practically unknown.

IV. Treatment.—Recovery can be looked for only at the onset of the disease. We should order good food, arsenite of potash and acetate of potash. Good results are also said to have been obtained from sulphur waters and baths.

b. Vesicle-like Inflammations of the Skin. Dermatitides Vesiculosæ.

1. Eczema.

I. Etiology.—The causes of eczema may be local or general. The local causes may be mechanical, thermal, or chemical.

The mechanical causes include obstinate scratching and rubbing the skin (in itching skin eruptions, such as prurigo, puritus, or urticaria, in scabies, wearing a rough hat, stiff collar, suspenders, etc.); constant friction of two opposed surfaces of the skin; finally, the eczema of the lower limbs produced by varicose veins.

Eczema caloricum often develops in the summer upon those parts of the integument which are exposed to the sun.

The group of chemical causes is very large. Thus, cutaneous applications of turpentine, croton oil, mustard, tartar emetic, mercurial and iodine preparations may give rise to eczema. In certain individuals eczema is produced even by the application of bland ointments. It is also observed in those who work in irritating substances (washerwomen, bakers, book-binders, mirror-polishers, etc.). It also occurs from wearing underclothing colored in poisonous dyes, particlurly arsenical aniline colors.

Among the eczemas of chemical origin must be included those produced by constant contact with perspiration, as in the axilla, inguinal folds, etc. It may develop on the lobe of the ear, as the result of discharge from the ear, on the eyelids, as the result of profuse lachrymation in conjunctivitis, on the upper lip after coryza, on the genitals after leucorrhœa. It is not infrequent upon the nates of infants who suffer from diarrhœa, and in whom the buttocks are for a long time in contact with the diarrhœal stools.

Symptomatic eczemas include those which are the result of internal diseases. There is danger, however, of mistaking an accidental complication as an etiological factor. Thus, the closeness of the relation between rickets or scrofula and eczema has been overestimated. It is known that patients suffering from Bright's disease and diabetes often suffer from obstinate eczema, and gastro-enteritic diseases and uterine affections often present the same complication. In some women, eczema occurs with a certain regularity at the menstrual period, in others it develops during each pregnancy. Violent mental excitement is occasionally mentioned as a cause.

Eczema is somewhat more frequent in the male sex. Age possesses little influence, except that the disease is rare before the age of six months. The influence of heredity is not proven, nor does the disease possess contagious properties.

Some individuals manifest a predisposition to eczema. It develops

in them after very trifling causes, and local causes produce it in a reflex manner in parts not directly attacked.

II. SYMPTOMS AND DIAGNOSIS.—The outbreak of eczema is preceded not infrequently by prodromata, particularly if the eruption is not the result of local causes. The patients feel chilly and weak, look pale and exhausted, and have slight fever. These symptoms generally subside with the outbreak of the eruption, but reappear at each relapse or exacerbation.

Eczema results in the production of an itching eruption, which is more often chronic than acute. It may appear in the form of closely aggregated, irregularly distributed, pale-red or dark-red papules (eczema papulosum), of small vesicles (eczema vesiculosum), or of pustules (eczema pustulosum). The skin may be red and moist (eczema rubrum et madidans); or the vesicles rupture, and their contents dry into thin, honey-yellow, rubber-like crusts (eczema crustosum); or the pustules are converted into thick, grayish-green or brownish-red crusts (eczema impetiginosum); or, finally, the integument is red, but does not weep, and is covered with thin, numerous scales (eczema squamosum).

All these forms are different stages of the same fundamental process. This is shown by the simple experiment of applying an irritant, such as croton oil, to the skin. Mild action of the oil produces erythema, at first hyperæmic, then exudative; stronger action results in eczema papulosum. If the irritant acts still more vigorously, serous, later purulent vesicles develop on the papules (eczema vesiculosum and pustulosum); when their contents dry, eczema crustosum and impetiginosum result. If the crusts fall off, a reddened, weeping cutis often appears (eczema rubrum et madidans). As the process heals, the weeping ceases, and fine scales form (eczema squamosum). If the irritant action is slight, eczema papulosum alone is produced. If the irritation is very intense, the preceding stages may be skipped, and eczema vesiculosum or pustulosum forms at once.

The various stages of development are sometimes found side by side in the same individual.

Eczema may be partial or general (rare), acute (duration, two to eight weeks), or chronic.

Eczema of the scalp (eczema capillitii) appears commonly in the form of impetiginous or squamous eczema. In the former we find thick, grayish-green or dirty-brown crusts, intimately adherent to the hairs. They are mixed with sebaceous secretion, and may diffuse a sour, rancid smell. If not kept clean, the scalp is apt to harbor lice, and, on the other hand, lice may give rise to impetiginous eczema. In such cases, the occiput and parietal regions often present prominences which, upon removal of the crusts, discharge green, creamy pus. The presence of pediculi is also shown by the nits on the hairs. In filthy individuals, the hairs may be matted together into an inextricable mass, containing crusts, innumerable lice and nits, and give rise to a pestilential stench (plica polonica).

Impetiginous eczema may be circumscribed or involve the entire scalp, and may also extend to the forehead, ear, and neck. The adjacent lymphatic glands are very often swollen.

In infants, impetiginous eczema of the scalp may be secondary to seborrhœa, the secretion decomposing and irritating the scalp.

If the crusts are removed, the corium appears reddened and weeping. When recovery takes place, the weeping ceases, and the reddened surface becomes covered with fine, white scales (eczema squamosum). In some cases, eczema squamosum develops as such. It is an annoying

and often obstinate affection, the scalp being always covered with more or less numerous white scales.

Eczema of the scalp is distinguished from seborrhœa by the fact that, in the latter, after removal of the crust the surface is not red or weeping, the glands are not swollen, and the disease does not extend beyond the scalp. It is distinguished from favus by the absence of spores in the crusts, and of the sulphur-yellow color, plate-like shape and peculiar smell of the favus crusts. Psoriasis of the scalp is associated with characteristic patches of psoriasis in other parts of the body.

Eczema faciei may extend over the entire face or be confined to certain parts. Diffuse eczema is not infrequent in infants beyond the age of five months. It generally appears as eczema crustosum or eczema rubrum et madidans. In the former, the face is covered with light-yellow crusts, which are often so closely aggregated as to form a sort of mask; in the latter, the face is red, moist, and sticky. The fluid is alkaline and very poor in cellular constituents. Eczema crustosum is often found in some parts of the face; eczema rubrum et madidans, in other parts which are on the way to recovery.

Eczema of the ear is apt to attack the posterior surface of the concha; it appears generally as eczema crustosum or impetiginosum. The concha is often very much swollen, and the swelling of the external auditory canal may interfere with audition. Eczema is often complicated with otorrhœa, or the former creeps along the external auditory canal. In some cases, the otorrhœa is primary, and gives rise to eczema. The latter often leaves behind deep and painful rhagades, which are sometimes covered with a gray, lardaceous coating, and emit a foul odor.

Eczema nasi affects the nostrils or the nasal mucous membrane. The nostrils are sometimes occluded by crusts, so that the patients are forced to breathe through the mouth. Snoring, stenotic sounds are produced on attempting to breathe through the nose. This condition is not devoid of danger in the new-born and nurslings, and may give rise to venous stasis, cyanosis, and pulmonary congestion. In not a few cases, moreover, the disease is the starting-point of facial erysipelas. If the eczema attacks the outer integument of the nose, very painful fissures may form in the naso-labial folds.

Eczema labiorum often gives rise to considerable swelling and deformity of the lips; they are thickly covered with brown or bloody crusts, and contain numerous painful, bleeding rhagades.

Eczema superciliorum and eczema palpebrarum produce great deformity. In the latter, the free border of the lid is not infrequently affected, and there may be secondary blepharitis.

Eczema barbæ (usually pustular or impetiginous) is still more annoying. Unlike sycosis, it very often extends beyond the beard. The two diseases are apt to be mistaken if the eczema extends to the hair follicles, and allows the hair to be readily removed with the tweezers.

Eczema nuchæ usually extends from the scalp. It often occurs independently in fat babies, in whom it develops between the horizontal folds of skin as the result of friction. If cleanliness is not enforced, these parts may undergo gangrene and ulceration, and, in rare cases, death has occurred in collapse or eclampsia.

Eczema mammæ is more frequent in women than in men. In stout women, eczema intertrigo develops not infrequently under the folds of the breast. In nursing women, eczema often occurs around the nipple,

and is frequently associated with very painful rhagades. Very severe eczema may extend to the mammary gland, and give rise to mastitis.

Eczema umbilici is observed particularly in obese individuals, and is extremely obstinate.

Eczema of the genitals occurs in both sexes. In males, it attacks the scrotum and dorsum penis, while the glans and inner surface of the prepuce escape. The scrotum is affected particularly in those parts which are in contact with the thighs. The disease may be associated with marked inflammatory œdema, so that the scrotum and penis are swollen and misshapen. After it has lasted a long time, the skin may undergo thickening and deform the genitals. The inflammation often extends to adjacent parts of the integument.

In women, the labia majora and mons veneris are most frequently affected, but the eczema sometimes extends to the labia minora and even to the vagina, giving rise to pruritus and secondary leucorrhœa. In other cases, leucorrhœa causes secondary eczema of the external genitals.

Eczema ani is annoying from the intolerable itching, and may give rise to painful rhagades, muco-purulent inflammation of the rectum, or prolapsus ani.

Eczema extremitatum is found not infrequently upon symmetrical parts of the limbs. In eczema of the folds of the elbow or the popliteal spaces, rhagades often form and interfere with the movements of the upper or lower limbs. The disturbances become still greater if the extensor and flexor surfaces are affected at the same time.

Eczema interdigitale is extremely annoying, and, if pustular in character, may be mistaken for scabies. Eczema digitorum sometimes affects only certain parts of the fingers; if the tips are attacked, the patients generally complain of formication in the fingers. The nails are sometimes involved, and may be exfoliated.

Eczema volæ manum often produces epidermoidal thickenings and deep-red, bleeding, or weeping rhagades. It may be mistaken for psoriasis, but in this affection patches of psoriasis are present in other parts. Eczema vesiculosum and pustulosum are also found here, but, as a rule, the thick epidermis interferes with the production of fully developed vesicles.

Eczema antibrachiorum appears not infrequently as a papular or vesicular variety. In the former, the skin is red, hot, and covered with numerous flat or pointed elevations; in the latter, it presents vesicles filled with fluid. Both forms occur not infrequently in harvest time as eczema caloricum. I have also observed them as the result of contact with rhus toxicodendron.

In *Eczema anticrurium*, the reddened skin feels sticky, and is covered with very fine, clear drops. The fluid stains the underclothing yellow, and stiffens it. In the process of recovery, there is often a transition to squamous eczema.

Eczema universale is rare. In many cases, the term may not be used literally, since some parts of the skin are intact.

In the majority of cases, eczemas disappear without leaving sequelæ. If very persistent, they may reduce the patient on account of the loss of appetite, and the disturbance of sleep caused by pruritus. Chronic eczema is sometimes followed by albuminuria; but, on the other hand, nephritis may also give rise to eczema.

III. ANATOMICAL CHANGES.—The same anatomical changes are

found as in the formation of papules, vesicles, and pustules in other eruptions.

In papular eczema are found: dilatation of the vessels in the upper layers of the cutis, serous exudation, emigration of white blood-globules, and swelling of the cells of the rete Malpighii. In vesicular eczema, the epidermis is separated in places from the rete Malpighii by the interposition of serous fluid. The vesicles are not infrequently multilocular, the septa being composed of flattened cells of the rete Malpighii, which have been pressed together. If numerous round cells enter the serous contents, pustular eczema develops. The pus-corpuscles are derived in part from the vessels of the cutis, in part from the cells of the rete Malpighii. A complete restitutio ad integrum may take place, but, in chronic cases, the cutis is unusually rich in cells, thickened, the blood-vessels and lymphatics are dilated, the sebaceous glands and hair follicles have partly disappeared, the sweat glands degenerated. Occasionally there is infiltration of the subcutaneous cellular tissue with round cells, and unusual pigmentation of the deeper layers of the rete Malpighii.

IV. Prognosis.—The prognosis, as regards complete and permanent recovery, is only unfavorable if the disease is the result of incurable constitutional causes.

V. Treatment.—The treatment depends mainly on the etiology. If the disease is the result of local injuries alone, local remedies alone are indicated.

In papular eczema, characterized by redness and heat of the skin, we may apply cool compresses of liq. alumin. acet. (1%), aqua plumbi or aqua plumbi Goulardi and water āā. To relieve violent itching, dust the parts at night with carbol-vaselin (1 : 15). If the pruritus is not severe, apply some dusting powder twice a day, for example, ℞ Flor. zinci, Sem. lycopod., āā ʒ i.; Amyli, ʒ iv. M. D. S.

As a general thing, vesicular and pustular eczema require treatment with bland oils or ointments, for example, the application of ol. olivæ, ol. amygdal., or unguent. simplex, etc.

In eczema crustosum or impetiginosum, the crusts must first be removed. This may be done by frequent application of the above-mentioned oils for two hours, or the application to the skin of a piece of flannel dipped in oil.

After the crusts are removed, the eczema is thus converted artificially into an eczema rubrum et madidans, and may be treated as such. In our opinion, the best ointment hitherto recommended in this disease is ung. diachylon Hebræ; if the itching is violent, it may be mixed with carbolic acid (1 : 50), if there is profuse weeping, with tannic acid (3 : 50). The ointment is smeared on pieces of old linen, and carefully applied with a flannel bandage. Unna impregnates mull with the ointment, and applies this directly as a bandage.

The number of ointments recommended in eczema is legion. Niemeyer extols white precipitate ointment as an almost sovereign remedy, and also obtained good results from corrosive sublimate (1 : 300). Lead and zinc ointments, ointments of nitrate of silver, subnitrate of bismuth, etc., are also recommended.

In squamous eczema, the skin should be anointed several times a day and strewn with the previously mentioned dusting powder. In very obstinate cases, tar preparations may be employed. Ol. rusci or ol. fagi et ol. oliv. āā may be applied once a day, so long as the scales are freely desquamated. Then the applications may be made at longer intervals,

but must be continued until the formation of scales ceases. In marked infiltration and thickening of the skin, good results are obtained from soap inunctions or applications of caustic potash (1 : 2 of water). The latter is applied once a week, and followed immediately by cold compresses to relieve the pain.

Indifferent baths and cold-water cures are useful in some cases.

If rhagades have formed behind the ear, salicylated cotton smeared with ointments may be applied. In eczema of the nose, tampons smeared with ointments should be introduced several times a day. In eczema of the scrotum, a suspensory bandage must be worn. In eczema of the rectum, suppositories containing oxide of zinc or tannic acid may be introduced.

The internal remedies include iron, iodine, and arsenic, according to the constitutional condition of the patient.

2. *Miliaria.*

Miliaria forms small, scattered vesicles which are transparent and clear (miliaria crystallina), or are surrounded by a red zone (miliaria rubra), or present a cloudy, almost purulent appearance (miliaria alba). They occur exclusively or chiefly on parts which are covered by clothing, and are almost always the result of increased production of sweat (sudamina).

The eruption appears in healthy individuals after profuse sweating in hot weather, or is produced artificially by sweat cures and cataplasms. It often develops during the crisis of acute febrile diseases. It is frequent in acute articular rheumatism, in the stage of recovery from typhoid fever, in phthisis, miliary tuberculosis, pyæmia, puerperal fever, measles, scarlatina, small-pox, the cold sweats of cholera, and even in agonal sweating.

A very trifling increase of cutaneous perspiration will produce sudamina in some individuals. On account of the varying irritability of the skin, miliaria crystallina persists as such, while in others, it is rapidly converted into the two other forms. Sometimes, indeed, miliaria crystallina remains almost entirely absent.

Miliaria crystallina forms clear vesicles, varying from the size of a millet seed to that of a pin's head; in rare cases a few attain the dimensions of a pea. When punctured, they discharge a clear fluid of a neutral or feebly alkaline reaction. The smallest vesicles are often better felt than seen. Sometimes there are thousands of vesicles on the skin, in other cases only a few. They sometimes disappear very rapidly. The fluid evaporates, the epidermis sinks in, forming small white elevations, or the vesicles rupture and the superficial layers of the epidermis are cast off. The eruption gives rise to no notable symptoms, at the most to slight pricking and itching. In some cases new outbreaks continue to form, so that the eruption lasts several days or weeks. In not a few cases it passes into

Miliaria rubra.—The contents of the vesicles become cloudy and a red zone forms around the periphery, as the result of decomposition of the vesicular contents and irritation of the deeper layers of the skin.

If the cloudiness increases and the red zone disappears, *miliaria alba* is the result. Both forms heal by desiccation of the contents of the ves-

icles and desquamation of the superficial epidermis. Treatment is un-
necessary.

Haight's examinations show that the vesicles are situated above the
excretory ducts of the sweat glands, and consist of elevation of the stra-
tum corneum. They are the result of occlusion or insufficient calibre of
the excretory ducts in question.

c. VESICULAR INFLAMMATION OF THE SKIN. DERMATITIS BULLOSA.

Pemphigus.

(*Pompholix.*)

I. SYMPTOMS.—The chief symptom of pemphigus is the occurrence
of vesicles upon the skin, varying from the size of a pea to that of the
palm of the hand or even more. The contents of the vesicle are, at first,
of a wine-yellow color, transparent, serous, and of neutral reaction; later
they become alkaline, cloudy, and pus like. The vesicles either rupture
spontaneously, laying bare the red weeping corium, which is gradually
covered with epidermis, and leaves behind a red, later brown patch; or
their contents dry into thin yellow, brown or blackened crusts which fall
off.

Closely aggregated vesicles are known as pemphigus confertus, isolated ones
as pemphigus disseminatus. In very rare cases only a single vesicle forms (pem-
phigus solitarius). If the vesicles are arranged in rows, the term pemphigus
gyratus is applied; if young vesicles form around an older one, pemphigus
circinatus. Vesicles whose contents have a bloody color are called pemphigus
hemorrhagicus.

Vesicles have also been observed upon the mucous membrane of the
lips, cheek, pharynx, nose, epiglottis, bronchi, vagina, gastro-intestinal
tract, and conjunctiva. The development of vesicles upon the epiglottis
may lead to fatal œdema of the glottis, and it is also claimed that pem-
phigus of the gastro-intestinal mucous membrane may be followed by
perforation.

Three varieties of pemphigus have been distinguished, viz.: pemphi-
gus acutus, chronicus, and foliaceus.

a. As a rule, acute pemphigus does not last more than a month. It
is more frequent in children than in adults; and may be epidemic in
maternity hospitals. As a rule, the first vesicles appear, in the new-
born, from the fourth to the ninth days. A number of cases of direct
contagion have been observed. It has also been found that when a large
number of cases occurred in a town, the children had been cared for by
a certain midwife. Inoculations with the contents of the vesicles, how-
ever, almost always are followed by negative results. Cases have been
observed in which the disease ran a febrile course and terminated fatally,
generally as the result of complication with pneumonia or gastro-enter-
itis.

Acute pemphigus may also occur in adults, but rarely runs an apyrex-
ial course. The eruption is often preceded for one to three days by pro-
dromata, viz., general malaise, anorexia, disturbed digestion, chill and
fever. The temperature increases with the appearance of the eruption,
and may recur at each new outbreak. We often notice erythematous
and urticaria-like places, upon which the vesicles form. The vesicle is
often surrounded by a red zone, gradually increases in size and becomes

tense, causes a disagreeable sensation of tension. It finally bursts or dries, while new ones form in other parts.

Vesicles in the mouth produce pain and fœtor ex ore; the buccal mucous membrane appears macerated in places. In pemphigus of the bronchi, membranous structures are sometimes expectorated (vide Vol. I., page 223).

Cardiac murmurs, enlargement of the spleen, occasionally albuminuria and hæmaturia have been observed as complications.

b. Chronic pemphigus lasts months and even years. In some cases the eruption continues uninterruptedly, in others there are periods of health followed by relapses. The disease may be entirely apyrexial, or there are temporary elevations of temperature. The patients may gradually grow very feeble, Bright's disease and waxy degeneration of the large abdominal glands may develop, and finally the patients may die from general marasmus. Stokes reported one case of exfoliation of all the nails following pemphigus of the fingers, and another case in which the vesicles appeared upon the scalp. A fatal termination is almost inevitable if the disease is converted into:

c. Pemphigus foliaceus. This form may also develop as such from the start, and Behrend described an epidemic of this kind in the newborn.

In pemphigus foliaceus, as a rule, the vesicles are small and flaccid, so that the epidermis upon them appears wrinkled. When the vesicle bursts, new skin does not form, so that new vesicles continually reenr, and the corium is laid bare more or less extensively. Hebra also observed a necrotic (diphtheritic) deposit upon the exposed corium. The disease almost always terminates fatally with the symptoms of increasing exhaustion.

II. ANATOMICAL CHANGES.—Recent vesicles are multilocular, the septa consisting of agglomerated scales of epidermis; older vesicles are unilocular. Upon the inner surface of the raised epidermis, there are often small appendages which correspond to follicles. The cells of the rete Malpighii are often broken up into granular masses, and the papillæ infiltrated with round cells.

In one case of pemphigus foliaceus J. Neumann observed dilatation of the sweat glands.

On perforation of the vesicles, the fluid almost always discharges readily, but Kuester reported a case in which some of the vesicles contained a gelatinus mass which was not discharged. The fluid contains changed white and red blood-globules, the remains of epithelium cells, granular detritus, and schizomycetes. In our opinion, however, the presence of the latter does not justify a conclusion as to the parasitic nature of the disease.

The chemical constitution of the fluid does not differ essentially from that of vesicles produced by a burn or a fly blister. Urinary analyses also give negative results.

III. ETIOLOGY.—The disease is more frequent in children than in adults, and in men than in women. Heredity has been occasionally noticed. It has been observed during pregnancy or the puerperal condition. Some cases are contagious in their origin. They may occur in infectious diseases, viz., pyæmia, puerperal fever, ulcerative endocarditis,

scarlatina, measles, small-pox, intermittent fever, and syphilis. Syphilitic pemphigus occurs almost always in children. It appears mainly, sometimes almost exclusively, on the soles of the feet and palms of the hand. The eruption is sometimes present at birth or in still-born babes. Toxic pemphigus sometimes follows the administration of potassium iodide or bromide, and salicylic acid.

IV. DIAGNOSIS.—The diagnosis is generally easy. It may be mistaken for malingering (action of blisters), burns, eczema, herpes iris, impetigo, urticaria bullosa, and erythema bullosum.

Baerensprung detected intentional fraud by finding, with the microscope, bits of the wings of the Spanish fly upon the vesicles.

The vesicles of burns are recognized by the previous history.

Eczema produces smaller vesicles, and the general condition is unaffected.

The differential diagnosis from herpes iris is extremely difficult at first, but is decided by the subsequent history.

In urticaria bullosa the itching is intolerable and wheals are also present.

In erythema bullosum we also find characteristic firm nodes which are not covered with vesicles.

V. PROGNOSIS.—This depends upon the nature and etiology of the disease. In pemiphigus foliaceus recovery is rare, in the acute form it is the rule. In chronic pemphigus the prognosis becomes serious if exhaustion and albuminuria make their appearance.

VI. TREATMENT.—Prophylaxis is important in some cases, particularly in pemphigus neonatorum (isolation of affected infants, avoidance of too hot baths, etc.).

Little can be expected from the administration of internal remedies. Iron, quinine, arsenic, iodine, acids, etc., have been tried.

If the patients complain of pain in the vesicles, the latter should be punctured and powdered with :

R Flor. zinci,
  Semin. lycopod......................................āā ℨ i.
  Amyli .... ℨ v.

M. D. S. To be used as a dusting powder; or the vesicles are covered with linen which has been smeared with carbolized oil, carbol-vaseline, etc. In addition, we may give a daily lukewarm bath (30° R.) containing ℥ x.–xvi. of soda, or a corrosive sublimate bath (ℨ i.–iij.) every other day. In pemphigus foliaceus permanent baths may be recommended.

In other respects symptomatic treatment.

d. PUSTULAR INFLAMMATIONS OF THE SKIN. DERMATITIDES PUSTULOSÆ.

1. *Impetigo and Ecthyma.*

I. SYMPTOMS AND ETIOLOGY.—The terms impetigo and ecthyma are applied to eruptions consisting of scattered pustules; secondary crusts are still more frequent than the pustules.

The term impetigo was formerly applied to pustules which do not exceed the size of a pea, ecthyma to larger ones, but there are such numerous transitions between the two that the distinction has been abandoned.

Hebra showed that in the large majority of cases, impetigo and ecthyma are symptoms of underlying processes.

Their causes may be local (idiopathic) or general (symptomatic).

The local causes, like those of eczema, are mechanical, thermal, or chemical (vide page 325).

Among the general causes are pyæmic processes, syphilis, glanders, and post-mortem wounds; also protracted, exhausting diseases, particularly typhoid fever.

Impetigo sometimes occurs in healthy persons without any demonstrable cause, lasts one to two weeks, is accompanid by fever, and then disappears entirely. The disease here develops idiopathically. This is also true of the contagious impetigo of Tilbury Fox, and the herpetiform impetigo of Hebra.

In contagious impetigo, pustules first appear upon the face, then upon the scalp, neck, trunk, and limbs, in a few cases upon the buccal mucous membrane and tonsils. The pustules dry up and form crusts, and the latter fall off without leaving a cicatrix.

The eruption is sometimes preceded by febrile prodromata, and the fever lasts several days after the outbreak of the eruption. As a rule, it recovers spontaneously by the end of the second week, often earlier, sometimes not until the sixth week. Unna observed several relapses.

A number of epidemics have been observed and several undoubted cases of infection. Several writers performed successful inoculations with the contents of the pustules, even upon the affected individual.

The contents of the pustules contain pus-corpuscles, epithelium cells and granular detritus. Micrococci have also been found. Kaposi observed higher fungi with fructification organs, and regards the disease as parasitic. Other writers could not confirm these results.

Impetigo herpetiformis was first observed by Hebra in five cases in pregnant women or soon after delivery. Recovery occurred only in one case. The following was the clinical history:

The eruption began on the inner surface of the thighs, and then extended to the anterior surface of the abdomen; the face and limbs, even the tongue, were sometimes attacked. Groups or circles of pustules first appeared and dried into crusts; these were followed by new erops in the vicinity. Upon removal of the crusts a reddened, weeping, but never ulcerated surface was exposed; it was sometimes covered with a smeary, grayish-white deposit, which occasionally emitted a disagreeable odor. Proliferations were sometimes observed upon the affected integument.

The eruption and also subsequent relapses were preceded by chills and elevation of temperature. Bloody diarrhœa was observed occasionally. Death occurred from increasing exhaustion.

II. DIAGNOSIS.—The diagnosis is easy. It is important to distinguish between ordinary and syphilitic impetigo. In the latter, other signs of syphilis are generally present, and, on removing the crusts, we find a deep, crater-like loss of substance covered with a grayish-yellow mass; in ordinary impetigo, the loss of substance is confined to the epidermis.

Contagious impetigo is distinguished from impetiginous eczema by the absence of itching.

III. PROGNOSIS.—This depends upon the etiology. In contagious impetigo, it is very good; in the herpetiform variety, it is almost always fatal.

IV. TREATMENT.—The treatment must first be directed against the

primary disease. In addition, the crusts should be thoroughly oiled, and, after their removal, the affected parts treated with bland ointments.

2. *Acne Vulgaris.*

(*Varus.*)

Acne vulgaris is an inflammation of the sebaceous glands, and in places of the hair follieles, which gives rise to the formation of red papules, nodes, and pustules. It is divided into five classes, viz., acne disseminata, frontalis, cachecticorum, syphilitica, and artificialis.

a. Acne disseminata is a very wide-spread affection. It is very rare in childhood, but almost always begins at puberty and often lasts for years.

The eruption is most profuse and constant on the forehead, chest, and back, rarer on the arms and thighs; the soles of the feet and palms of the hand always escape (absence of sebaceous glands). Arlt observed acne of the conjunctiva.

Seborrhœa and the formation of comedos are generally observed at the same time. Indeed, the latter gives rise in many places to acne, because the stasis produced by the comedo results in inflammatory irritation. In the mildest form we find little red papules, in the middle of which is a black comedo (acne punctata), in other places a pustule has formed upon the nodules (acne pustulosa). The inflammation extends not infrequently to the adjacent cutis, and forms red, very painful nodes, from the size of a pea to that of a bean (acne indurata); in many cases, pus does not escape until the scalpel is introduced very deeply. The papules, nodes, pustules, and comedos may be so numerous as to cause great deformity. Exacerbations and remissions are noticeable.

The nature of the etiological relation between puberty and acne is entirely unknown.

On microscopical examination, the excretory portion of the sebaceous follicle is found occluded by thickened sebum, the gland itself is dilated and filled with fat granules, cellular detritus, and pus-corpuscles. The adjacent cutis is congested, and more or less infiltrated with round cells. If lateral pressure is exercised upon the acne, the plug of sebum with its black head first appears, and is followed by milky or purulent, pasty or more fluid contents.

b. Acne frontalis is distinguished from acne disseminata, as regards its mode of development, by the fact that it is not preceded by the formation of comedos. It occurs exclusively on the forehead, particularly near the hair. The papules or pustules are flat, and often carry a small, central crust. This is deeper than the periphery of the nodule, so that the latter forms a sort of wall. When the crust drops off, a slightly depressed cicatrix remains.

c. Acne cachecticorum develops after long protracted and exhausting diseases; it is associated not infrequently with pityriasis tabescentium and lichen scrophulosorum. It is probably produced by hyperplasia of the epithelium of the sebaceous follicles, as the result of cachexia with subsequent inflammatory irritation.

d. Acne syphilitica (vide article on Syphilis, Vol. IV.).

e. Acne artificialis. The best known form is tar acne, produced by local applications or by inhalations of tar vapor. If the skin is coated with tar, black specks (particles of tar) are observed in the sebaceous

follicles, and later the acne changes appear. If tar is inhaled, it is probably deposited in part in the sebaceous follicles, and there produces inflammation. Tar acne is also produced in those who work in tar products (creasote, benzin, petroleum, etc.). Acne also develops from the use of iodine and bromine preparations and chrysarobin ointment.

The diagnosis of all the varieties of acne is easy, the prognosis is unfavorable so far as regards thorough and permanent cure.

In acne cachecticorum, reliance must be placed on cod-liver oil, tonics, and nutritious food; local treatment is hardly necessary.

In acne disseminata, we should first attempt to prevent the formation of comedos. If there is an abundant formation of papules or nodes, mercurial ointment should be smeared on leather and applied at night to the affected parts. The next morning the skin is carefully washed with the following: ℞ Saponis virid., ℥ iij., Spiritus vini, ℥ iv.; filtra et adde Ol. lavandul., Ol. bergamotæ, āā gtt. v. M. D. S. To be used externally. Large, painful nodes should be poulticed, and then incised. If there is a profuse formation of pustules, these should be punctured, and their contents discharged; the skin may then be covered with Hebra's diachylon plaster.

We may briefly mention the following other plans of treatment: *a.* Applications of corrosive sublimate (gr. ¾ to iss. : ℥ iiiss.); *b.* application of tinct. iodin., tinct. benzoes, or tinct. cantharid.; *c.* treatment with sulphur or glycerin soaps; *d.* (Zeissl's sulphur paste) Lac. sulph., Kali. carbon., Glycerin., Spirit. vin. dilut., āā. M. D. S. Smeared on leather, and applied every night, removed with soap in the morning, etc. All these remedies seek to produce active desquamation of the superficial layers of epidermis, and thus to keep the excretory ducts of the glands free.

3. *Acne Mentagra.*

(*Sycosis.*)

I. ETIOLOGY.—The term sycosis is applied to a chronic inflammatory process in the hair follicles, which gives rise to the formation of papules, nodes, diffuse infiltrations of the skin, pustules, and crusts. Two forms are distinguished, the parasitic (produced by tricophyton tonsurans), and the non-parasitic.

The disease is observed almost exclusively in bearded men. A dense growth and very thick hairs in the beard predispose to sycosis.

The causes of non-parasitic sycosis are unknown. Among those which have been assumed are shaving with a dull razor, uncleanliness, irritation by snuff, highly spiced food. In some cases, it follows eczema. Chronic coryza may also give rise to sycosis if the secretion constantly flows upon the upper lip, and irritates the follicles.

Hebra believes that in sycosis a new hair grows in an old hair follicle before the previous one has fallen out, and thus produces mechanical irritation and inflammation of the hair follicle. Werthheim assumes that the beard hairs are too thick, so that the follicle does not afford sufficient room for the transverse section of the hair.

Parasitic sycosis is produced by direct infection with herpes tonsurans, either from animals (cow, horse, dog) or from man to man.

The disease affects the head most frequently, and has therefore been called folliculitis barbæ. More rarely it attacks the eyebrows or lids, still less frequently the hair of the axilla, mons veneris, the vibrissæ of

the nose, or the scalp. In the latter locality, the disease is almost always secondary to eczema.

II. SYMPTOMS.—The development of the eruption is generally preceded by a feeling of tension and pricking in the affected parts. Then there appear red papules and nodes, whose centre is perforated by a hair. Adjacent nodes come in contact, and form diffuse nodular elevations; not infrequently there are extensive infiltrations of the skin. Pustules appear in places upon the nodes, and either rupture spontaneously or dry into yellow or grayish-yellow crusts, which are likewise perforated by a hair. In those localities in which a hair is situated in the pustule,

FIG. 135.

Hair containing fungi from a node of parasitic sycosis. Half of the hair alone is drawn. After Kaposi.

the former is readily removed on making traction. The root portion appears thickened, and the root sheaths infiltrated with pus; the hair is often bent at an angle immediately above the bulb. In parasitic sycosis, the hair is peculiarly dry, fibrillated, and discolored. After removal of the hair, a little drop of creamy pus often escapes.

If the crusts are softened and removed, we often find reddened but dry surfaces, which are sometimes elevated like condylomata, present the appearance of proud flesh, or are perforated like a sieve, the individual openings containing pus. After the process has lasted for some time, certain parts of the skin may be entirely destitute of hair. This condition persists, inasmuch as the hair follicles have been obliterated by

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the inflammation. The submaxillary lymphatic glands become swollen if the cutaneous changes are extensive.

In non-parasitic sycosis, the changes described are confined strictly to the beard; in the parasitic form, they are apt to extend to adjacent parts of the face or neck. The changes characteristic of herpes tonsurans make their appearance in sharply defined circles, which are covered with little vesicles or scales.

Non-parasitic sycosis may last a very long time, sometimes even more than thirty years. Parasitic sycosis generally runs a rapid course.

III. DIAGNOSIS.—The diagnosis is easy. Sycosis is distinguished from eczema by the fact that in the latter the affected skin is moist and itching, and that it is not confined to the hairy parts. The microscope readily distinguishes parasitic from non-parasitic sycosis; in the former, the hairs contain threads and spores of tricophyton tonsurans (vide Fig. 135).

The fungus is formed earliest between the hair and inner root sheath, then it spreads into the root sheaths, finally into the substance of the hair. The hairs become transparent if treated for fifteen to twenty minutes with potash (1 : 3). Among four hundred examinations, Michelson and Schueppel found the fungus only in one hair out of twenty.

IV. PROGNOSIS.—The prognosis is good. The disease may recover spontaneously, and is amenable to treatment, but it exhibits a tendency to relapse.

V. TREATMENT.—In non-parasitic sycosis, the beard should be closely clipped if the disease is extensive. The beard should be shaved daily, even long after the disease has recovered, in order to prevent a relapse. Crusts are removed by fatty inunctions (two hours), pustules are opened, and the hairs contained therein are removed daily with a pair of tweezers. Only one hair should be grasped at a time and traction made in the direction of the hair. The entire diseased district should not be treated in the first few sittings, since this may give rise to peculiar conditions of excitement, and even to syncope. Epilation must be continued until the formation of nodes and pustules has ceased. Behrend has recently employed the sacrificator with success. The remaining infiltration may be treated with Hebra's diachylon plaster or mercurial ointment; if the infiltration is very thick, with Zeissl's sulphur paste.

Parasitic sycosis may be treated with applications of turpentine, corrosive sublimate, nitrate of silver, acetic acid, or powdered sulphur.

APPENDIX.

Hebra applied the term sycosis framboesiformis to an affection located at the border of the neck and hair, and which gives rise at first to the development of nodes, each of which is perforated by a hair. Later, the coalescence of the nodes causes a raspberry-like infiltration of the hairs, and which, in many cases, cannot be removed except by cauterization or excision. The disease is chronic; it is perhaps the result of mechanical causes (friction of the collar.'

4. *Acne Rosacea.*

I. SYMPTOMS AND ANATOMICAL CHANGES.—The disease occurs exclusively upon non-hairy portions of the face, generally the nose, next the cheeks, chin, or glabella. It may also extend continuously over

large portions of the face, sometimes to the border of the hair, and even to the back of the neck.

In the mildest grades, there is unusual redness of the skin, partly diffuse, partly in the form of unusually dilated and sinuons cutaneous vessels. It usually increases after eating or excitement. It is often associated with seborrhœa.

As the disease grows more severe, soft, painless, red nodules, varying from the size of a lentil to that of a pea, appear upon the reddened skin. In addition, there is often an abundant development of comedos and acne vulgaris. Pustules are rarely observed.

The process is the result of marked dilatation and in part of new-formation of vessels of the cutis, dilatation of the sebaceous glands, with stagnation of their secretion, and the formation of a gelatinous connective tissue in the cutis. Spontaneous recovery is possible, even in this stage, by absorption of the newly formed connective tissue. In the majority of cases, however, the process continues to advance.

FIG. 136.

Large nodes then develop, generally situated on a broad base, more rarely pedunculated. If the disease attacks the nose, great deformity is produced; the organ sometimes attains the size of two fists, and looks like a large rough potato, etc. (rhinophyma).

II. ETIOLOGY.—In many cases, the disease is the result of alcoholic excesses.

The greatest danger is run by wine drinkers, next by brandy drinkers, finally by beer drinkers. Those wines which are rich in ethereal oils and acid are the most injurious in their effects. In whiskey drinkers, the nose is usually smooth and bluish-red; in wine bibbers it is generally covered with red protuberances; in beer drinkers, with cyanotic tubercles.

Acne rosacea is sometimes found in individuals who suffer from diseases of the digestive tract (stomach, intestines, liver).

In women, it is almost always the result of diseases of the sexual apparatus: amenorrhœa, uterine diseases. It is most frequent in females at the beginning of puberty or the menopause; in males, it develops beyond the age of 35 years. In some women, the first signs appear during pregnancy or childbed. It has been often observed after protracted cold-water cures.

Finally, the disease may follow local injuries (frequent exposure to rough winds or to fire).

Lancet-shaped needle fo scarification.

III. DIAGNOSIS.—The diagnosis of the disease is easy. It may possibly be mistaken for frost-bites, lupus, carcinoma, syphilis, and rhinoscleroma.

In frost-bites, the skin is bluish-red, and the cutaneous veins are not dilated.

Lupus erythematosus is characterized by profuse formation of scales and the development of cicatrices.

In lupus vulgaris, there are brownish-red nodules, which are apt to break down and cicatrize.

Carcinoma, as a rule, soon ulcerates. This is also true of syphilis; in addition, other evidences of syphilis are noticeable.

IV. PROGNOSIS.—The disease is often very obstinate. The de-

formity is often so great that the patients are a laughing-stock in the community.

V. TREATMENT.—We must first endeavor to remove the cause of the disease, and this in itself is sometimes sufficient to effect a cure. In addition, local treatment must be adopted.

In the mildest form (simple redness), we recommend that the skin be brushed every night with collodion or sublimate collodion (gr. $\frac{3}{4}$: $\frac{7}{3}$ i.). We have also obtained good results from an ointment of tannin and ergotin (ung. diach. Hebræ, 3 v., acid. tannic., ergotin., āā 3 ss. M. D. S. To be smeared on linen, and applied every night).

The second grade of the disease may be treated with mercurial ointment and, if there is marked vascularization, by scarification with a lancet-shaped needle (Fig. 136). Zeissel's sulphur paste, iodine and sulphur preparations may be employed to relieve marked infiltrations.

Cases of the third grade must be treated by surgical interference, *i. e.*, removal of the new-formations.

e. SCALE-FORMING INFLAMMATIONS OF THE SKIN. DERMATITIDES SQUAMOSÆ.

1. *Psoriasis.*

I. SYMPTOMS.—Psoriasis is a chronic skin disease which leads to the production of abundant scales possessing a gloss like that of mother-of-pearl. If these are removed with the nail, a reddened, easily bleeding base makes its appearance.

The individual efflorescences begin as a reddened, slightly elevated patch, which is covered, at the end of a few days, with an increasing number of scales, and, at the same time, increases in size. The smallest efflorescences form little dots which shine like asbestos (psoriasis punctata); when they attain the size of a lentil, the skin looks as if sprinkled with mortar (psoriasis guttata). In some places, the scales disappear in the centre of the patch, but extend further at the periphery (psoriasis orbicularis). Adjacent patches not infrequently coalesce and produce complicated figures (psoriasis gyrata). If the scales extend continuously over a large area, it is known as psoriasis diffusa. The term psoriasis nigra is employed when the scales have a brownish or blackish appearance (particularly in old cases).

The eruption first appears on the extensor side of the elbows and knees, and these parts are rarely unaffected in extensive psoriasis. As a rule, the scalp is next affected, thick hillocks of scales being formed and matting the hairs together. At the border of the hair, the patches often extend to the adjacent integument of the face and neck. The concha and external auditory canal are very often affected, but there are many cases in which the eruption extends over the entire body. The mucous membranes are always intact. The volar surfaces of the hands and feet are rarely affected, in contradistinction to psoriasis syphilitica. Psoriasis sometimes develops on the nails; white patches first appear, then the nail thickens, becomes lamellated, and crumbles to pieces.

In some patients, a few patches persist, perhaps for life; in others, there may be very little unchanged integument. Remissions and exacerbations occur very often. The eruption sometimes disappears spontaneously, and remains absent for months and years. As a rule, the cause of the relapse cannot be ascertained.

Very violent exacerbations may be associated with slight fever, pain in the joints, and neuralgic symptoms. The patients often suffer no subjective disturbances, but at first some complain of itching. If the psoriasis is very marked at the large joints, very painful rhagades develop not infrequently, and render motion distressing. In inveterate psoriasis of the face, we also find painful fissures, occasionally also ectropium. As a rule, individuals suffering from extensive psoriasis do not perspire much. Cutaneous sensibility is sometimes diminished, and albuminuria has also been observed. If the disease is very obstinate and extensive, the patient may suffer from anorexia, with increasing feebleness and exhausting diarrhœa, and these symptoms occasionally terminate fatally.

If spontaneous recovery ensues, the scales fall off, the skin is at first red and infiltrated, but gradually grows pale and returns to the normal. In some cases, pigmented spots persist upon the legs.

Fig. 137.
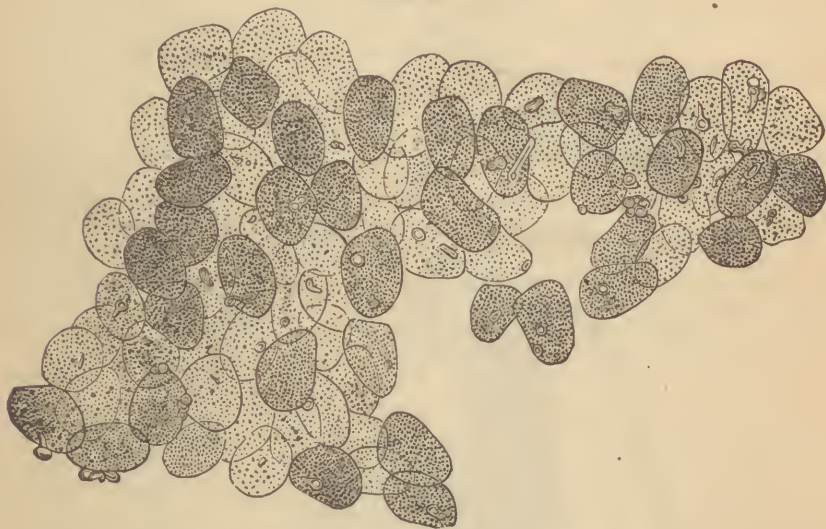
Spores from the psoriasis membrane. After Lang.

ETIOLOGY.—Little is known concerning the causes of the disease. Heredity exercises an important influence; the inheritance is sometimes direct from the parents, sometimes from the grandparents or collateral branches of the family. The predisposition to psoriasis alone is inherited, and the development of the disease requires the interposition of certain external irritants.

The latter include traumatism. Hence the early appearance of psoriasis on the elbows or knees, because these parts are particularly subject to friction. J. Neumann showed that the disease not infrequently follows eczema intertrigo, the latter being secondary to traumatism.

All other statements with regard to the etiology of psoriasis are either improbable or unproven.

Next to eczema, psoriasis is the most frequent of skin diseases. As a rule, it does not begin before the sixth year, nor after the fortieth year. It seems to be somewhat more frequent in men than in women.

According to a few statements which have been made, psoriasis is contagious.

Lang recently found a fungus (epidermidophyton) in this disease, so that h classes it among the dermatomycoses. Very few writers have succeeded in verifying Lang's statements. The following is a brief description of his method and results.

If the scales upon a patch of psoriasis are carefully removed, a thin, transparent, little membrane is brought into view. This is quickly removed with a pair

Fig. 138.

Fungus threads from the psoriasis membrane. After Lang.

of forceps, and placed in a five-per-cent solution of potash. After the scales have swollen and become transparent (at the end of a few minutes), we will find between them, round, oval, and elongated spores, 0.006 to 0.008 mm. broad and twice as long (vide Fig. 137). If the membrane is placed in a solution of potash which has been mixed with equal parts of glycerin and water, the spores will develop, at the end of two or three hours, into long, rarely branched threads, whose free end is either rounded or swollen (vide Fig. 138).

III. ANATOMICAL CHANGES.—Since some of the lesions disappear after death, sections must be taken from the living subject.

In the cutis are observed elongation and increased breadth of the pa-

pillæ, dilatation and sinuosity of the vessels, profuse emigration of white blood-globules, and accumulation of the latter, particularly upon the outer wall of the vessels. J. Neumann also found the sweat glands filled with round cells. In the rete Malpighii the lowermost palisade-like epithelium cells, which are arranged normally in one row, now occupy several rows; the higher epithelium cells have lost their processes, and also present nuclear proliferation and fission.

IV. Diagnosis.—The disease possibly may be mistaken for eczema squamosum, seborrhœa capillitii, pityriasis rubra, pemphigus foliaceus, lichen ruber. lupus erythematosus, lupus exfoliativus, rupia, psoriasis syphilitica, favus, and herpes tonsurans.

Eczema squamosum presents marked pruritus, often weeps, and is preceded by the formation of vesicles.

Seborrhœa capillitii occurs particularly in the new-born, never extends beyond the border of the hair, and, upon removal of the crusts, a red, bleeding surface is not exposed.

Pityriasis rubra is distinguished from psoriasis by its course; it leads to marasmus and serious general disturbances. This is also true of pemphigus foliaceus, in which, moreover, vesicles are formed.

In lichen ruber, the efflorescences are smaller, covered with fewer scales, and, as a rule, are arranged in contiguous groups.

Lupus erythematosus develops chiefly on the nose and cheeks; on removal of the scales, their lower surface is found to present prolongations which project into the sebaceous follicles; after recovery from lupus, cicatrices and pigmentations are left over.

Lupus exfoliativus produces fewer scales, and on their removal the surface is found reddened, but not bleeding.

After removal of a rupia crust, a deep, crater-shaped ulcer appears, with a lardaceous base.

Psoriasis syphilitica is attended with a slighter formation of scales, which adhere more firmly, the base is brownish-red; it is localized on the palms of the hands and soles of the feet, and other evidences of syphilis are present.

Favus and herpes tonsurans are recognized with the aid of the microscope by the presence of the characteristic fungi.

V. Prognosis.—The prognosis is almost always good as regards life, but is unfavorable as regards complete recovery, since it is impossible to prevent relapses.

VI. Treatment.—Internal treatment should be combined with external treatment.

We have succeeded in almost every case with the following simple plan. The patient receives:

℞ Aq. amygdal. amar.,
    Liq. potass. arsenit...................................āā ℥ ss.
M. D. S. Five drops t. i. d. after meals; the dose is increased by one drop every third day.

In addition, the patient should take a daily bath at 30° R. (lasting two hours), in which ℥ iij.–vi. of potassium sulphate are dissolved. Immediately after the bath, the following ointment is rubbed into the patches with a brush, until the scales are removed as thoroughly as possible.

℞ Sapo. virid.,
    Picis liquidæ,
    Lact. sulphur.,
    Spirit. vini dilut.................................āā ℥ i.
M. D. S. To be used externally.

We will mention a few other remedies which have been employed in this disease. *a*, Internal remedies. Iodine and mercurial preparations, carbolic acid, salicylic acid, tar preparations, copaiba, carbonate of ammonia, phosphorus, etc.; also exclusive meat diet. *b*, External remedies. Subcutaneous injections of arsenical preparations, douches, hydropathic packs, permanent baths, cures ·n Aix, Kreuznach, Pfaffers, etc., preparations of tar, iodine, and sulphur, cauterization with acetic acid or corrosive sublimate, chrysarobin (℥ ij. ; vaselin ℥ x.), pyrogallic acid (℥ i. : x.), thymol, iodoform.

2. *Pityriasis Rubra.*

I. SYMPTOMS.—This is a very rare disease. The skin reddens and desquamates without any previous effloresence. Later the skin grows thinner, is tense, and appears to have become too small for the body. The face assumes a stiff, mask-like expression, the limbs are in a forced position, midway between flexion and extension. Rhagades form here and there. Alopecia often develops at a later period. The disease sometimes starts from a certain part, sometimes it is diffuse from the beginning; at all events, it tends to spread over the entire body.

The disease is almost always chronic, and may last several years. Gangrene of certain parts has been observed. At the same time marasmus sets in, and terminates fatally.

II. DIAGNOSIS.—It is distinguished from squamous eczema by the absence of moisture of the skin and of marked itching. In lichen ruber, the formation of scales is attended with the development of nodules; lupus erythematosus occurs chiefly in the face.

III. ANATOMICAL CHANGES.—Hebra found, at the onset, profuse infiltration of the upper layers of the cutis with round cells, later atrophy of the epidermis and papillary body, atrophy of the sweat glands and sebaceous glands, and sclerosis of the cutis tissue. Phthisical changes have been observed in the lungs and intestines.

IV. ETIOLOGY.—The causes and nature of the disease are unknown.

V. PROGNOSIS.—The prognosis was formerly regarded as absolutely fatal, but some cases of recovery have been recently reported.

VI. TREATMENT.—Internal administration of arsenic or carbolic acid, externally baths and fatty inunctions.

f. PAPULAR INFLAMMATIONS OF THE SKIN. DERMATITIDES PAPULOSÆ.

1. *Prurigo.*

I. SYMPTOMS.—Prurigo is characterized by the appearance of scattered papules, from the size of a pin's head to that of a hempseed, which either possess the normal color of the skin, or are pale red. On puncture, they discharge a clear serous fluid. The disease is chronic, and is attended with intolerable itching.

The first symptoms often appear in childhood, generally towards the end of the first year, and may then last for the remainder of life. They often appear as an obstinate, relapsing urticaria. The characteristic prurigo papules gradually develop under the epidermis, so that they are better felt than seen. Later the papules project more and more above the level of the skin.

They appear first and most abundantly upon the legs, then upon the thighs, forearms, arms, and trunk. A few also appear upon the face. Upon the scalp we only find vigorous branny desquamation of the skin, and dryness and falling out of the hairs. The popliteal space, elbows, axillæ, palms of the hands, soles of the feet, and genitalia always escape.

The intolerable itching is most severe at night, especially when the patients are warm in bed. Secondary efflorescences develop as the result of the mechanical irritation by scratching. The apex of the

scratched papule acquires a bloody crust, or is converted into a pustule. Pustules, eczema, and urticaria also develop in places which are free from prurigo papules. The lymphatic glands become swollen, particularly in the groins, where they may form masses as large as a pigeon's egg. These are somewhat characteristic of the disease, and are known as prurigo buboes. The glands rarely suppurate. After the disease has lasted a long time, the skin assumes a brown color (melasma), and feels infiltrated and thickened. The furrows of the skin are not infrequently unusually deep. The skin is generally dry, produces but little perspiration, and is often covered with scales. Warty elevations are sometimes found upon the legs, and the muscular system may undergo considerable emaciation. The general condition may suffer from disturbed sleep.

Remissions and exacerbations are frequent. The symptoms increase during the winter, and may disappear almost entirely during the summer.

II. ETIOLOGY.—The causes of the disease are unknown. Heredity seems to exert an influence in some cases, but since prurigo does not develop before the end of the first year, we must assume that merely the predisposition of the skin to prurigo is developed. It has been attributed to highly spiced diet, scrofula, rickets and phthisis (?). It is more frequent in males than in females, and also among the children of the poorer classes.

III. ANATOMICAL CHANGES.—Hebra attached chief importance to the collection of a fluid substance between the cells of the rete Malpighii, which served to nourish the cells, but was secreted in excessive amounts. J. Neumann noticed cell proliferation in the papillæ, and swelling of these bodies by inflammatory, purely serous exudation.

Derby states that each papule is perforated by a hair. He also described hypertrophy of the arrector pili, increased development of the external root sheath and bulbous involutions of the hair follicles. These changes are of minor importance. Gay noticed dilatation of the lymph spaces in the cutis. The following changes have also been described: Proliferation of the cells of the rete Malpighii, proliferating processes in the sweat glands, in old cases atrophy of the sebaceous and sweat glands, and abnormal deposit of pigment in the layers of the cutis.

Schwimmer claims that prurigo is a trophoneurosis. In our opinion, however, the pruritus is secondary to the development of the prurigo papules.

IV. DIAGNOSIS.—The diagnosis is easy, if we bear in mind the characteristic shape and distribution of the efflorescences. We must be careful to avoid regarding secondary cutaneous changes as the primary lesion.

V. PROGNOSIS.—The disease is capable of recovery if treated early. Permanent recovery is not infrequent in children. But if the disease has lasted more than four years, complete recovery will hardly ever be obtained.

VI. TREATMENT.—Much cannot be expected from the use of internal remedies. Arsenic and carbolic acid have been particularly recommended. The general condition should also be treated.

The following is the simplest and most certain plan of local treatment: Every night the patient is rubbed with green soap; in the morning he takes a bath (for two hours) at 30° R., ℥ iij.–vi. of potassium sulphate being added to each bath. Then the entire skin is anointed

with carbol-vaseline (3 : 50). This plan must be continued for a long time, even after the papuleˢ have disappeared.

Good effects have been obtained from subcutaneous injections of pilocarpine or carbolic acid. The treatment recommended for psoriasis is also much in vogue for prurigo.

2. *Lichen Scrophulosorum.*

I. SYMPTOMS.—In this disease are found papules about the size of a pin's head, of a pale red, livid red or brown-red color, with a little scale upon the apices. If the latter is removed with the nail, the mouth of a hair follicle appears, the vicinity of which is slightly raised like a wall. These papules are generally arranged in round groups, rarely in circular lines. They itch slightly or not at all, and disappear spontaneously after branny desquamation. The disease is chronic, and often lasts for years.

The first efflorescences appear upon the back, chest, and abdomen, later upon the limbs, particularly the flexor surfaces; finally, the face and scalp may be affected. In advanced cases, large areas may be involved. Acne and eczema may also be present, the latter particularly on the genitalia.

II. ETIOLOGY.—The disease is most common in children, especially in boys. It is rare beyond the age of 20 years. The patients generally present the evidences of scrofula. The skin is often very pale, and is peculiarly fatty to the feel. In exceptional cases, phthisical changes are noticed.

III. ANATOMICAL CHANGES.—Kaposi found infiltration of the cutis with round cells in the immediate vicinity of the hair follicle and its sebaceous glands, and in the papillæ immediately adjacent to the follicle, round cells in the follicles themselves, and accumulation of epidermis cells in the mouth of the hair follicle.

IV. DIAGNOSIS.—The diagnosis is easy. In squamous eczema, there is severe itching, and many papules are converted into vesicles and pustules. Lichen syphiliticus affects chiefly the flexor surfaces of the limbs, and the papules are often as large as a pea.

V. PROGNOSIS.—The prognosis is good, since there is no danger to life and the disease is not incurable.

VI. TREATMENT.—This consists of the internal and external administration of cod-liver oil, one tablespoonful internally morning and evening, inunctions externally t. i. d., and the parts then covered with flannel.

3. *Lichen Ruber.*

I. SYMPTOMS.—This extremely rare disease appears as scattered papules, as large as a pin's head, of a pale red or brownish-red color; they are sometimes pointed and covered with thin scales, sometimes flat, and appear umbilicated. Both forms are very often combined.

The first papules appear on the chest, abdomen, genitalia, or flexor surfaces of the limbs. New ones gradually appear and finally they are so closely aggregated as to come in contact. In the course of years, almost the entire integument may be attacked. The scalp, axillæ, and mons veneris are generally unaffected, the hairs of other parts fall out.

Peripheral circles of papules sometimes sprout up around central

ones, or the latter disappear, leaving behind pigmentation and cicatricial changes, while the process extends at the periphery.

In old cases, the palms of the hands and soles of the feet are covered with thickened, fissured epidermis, the nails are also thickened, brittle, brownish, or thinned. The papules have also been observed on the mucous membrane of the mouth. The red, scaly skin is often diffusely infiltrated, and painful, bleeding rhagades are formed; the limbs are kept semi-flexed, and movement is painful.

If the process is left to itself, increasing marasmus sets in, and finally death. In some cases sleep is disturbed by pruritus, and this accelerates the collapse.

II. ETIOLOGY.—The disease is more frequent in men. It develops mainly between the ages of ten and forty years, rarely earlier, though Kaposi reports a case in a child of eight months. It causes are unknown.

III. ANATOMICAL CHANGES.—Examination of the skin shows nothing which has not also been observed in other chronic skin diseases. We may mention infiltration of the papillæ with round cells ; obliteration, sometimes dilatation of blood-vessels in the papillæ ; occasionally colloid degeneration of the walls of the vessels. In umbilicated papules, the papillæ corresponding to the umbilication are atrophied; the external root sheath of the hair follicles is unusually well developed; hypertrophy of the arrectores pili; fibrillation of the lower end of the hairs; increase of the cells in the rete Malpighii and epidermis.

IV. DIAGNOSIS.—The diagnosis is generally easy. The disease is distinguished from papular and squamous eczema by the fact that vesicles and pustules have generally been present in the latter affections. Psoriasis forms thicker scales, and the patches increase peripherally, while lichen papules always retain the same size. Lichen ruber is distinguished from lichen scrophulosorum by the absence of a tubercular etiology, and by the different distribution of the eruption.

V. PROGNOSIS, TREATMENT.—The prognosis is good, since Hebra found that successful results are obtained by the internal or subcutaneous administration of arsenic In marantic individuals, good diet and iron preparations are important. If there is violent itching, the skin should be anointed night and morning with carbol-vaseline (3 : 30). Instead of arsenic, Unna recently recommended the following ointment: Unguent. zinc. benzoat., ℥ x., Acid. carbolic., ʒ iij., Hydrarg. bichlorid., gr. iv. M. D. S. To be rubbed into the skin morning and evening. The patient then remains between woollen blankets.

PART II.

SECRETORY ANOMALIES OF THE SKIN.

A. Secretory Anomalies of the Sweat Glands.

I. *Increased Secretion of Sweat. Hyperhidrosis.*

(*Ephidrosis.*)

Unusually profuse production of sweat may affect the entire integument (hyperhidrosis universalis) or be confined to certain regions (hyperhidrosis localis).

Hyperhidrosis universalis is often observed in well-nourished, obese individuals who perspire profusely on slight exertion. This perspiration is most abundant in inclosed portions of the body (axillæ, anal groove, etc.). The excessive perspiration often produces further cutaneous changes. At first sudamina develop (vide page 330). In other cases, the skin appears macerated and slightly reddened, especially where two surfaces rub against one another (chafing, intertrigo).

In epileptics, profuse diaphoresis has sometimes been observed, either in the place of the epileptic attacks, or shortly preceding them.

We will not consider the diaphoresis observed during the course of internal diseases, since this is a symptom of the diseases in question.

Hyperhidrosis localis may be unilateral or circumscribed still more narrowly.

Unilateral hyperhidrosis has been observed in the course of various nervous diseases (idiocy, Basedow's disease, asthma). Pikroffsky reports a case in which the ingestion of food always produced sweating on the right side of the body. In Kaposi's case, it occurred upon the face on one side, and the trunk and limbs on the opposite side.

In some cases, unilateral sweating is confined to the face. This has been observed in general paralysis of the insane, in phthisis with large cavities (the sweating side corresponded to the side of the cavity). In Donder's case, it always occurred during mastication.

In one case, Riehl found that the superior ganglion of the cervical sympathetic on the affected side was swollen and reddened, infiltrated with round cells, the vessels distended, the ganglion cells and nerve fibres atrophied, and containing a punctate hemorrhage.

Hyperhidrosis localis is sometimes very circumscribed. In one case, diaphoresis occurred, at certain hours of the day, upon the dorsal surface of the right hand and forearm. This was relieved by quinine.

Excessive sweating in the axillæ not infrequently stains the clothing of a yellowish-red color; in addition it produces a disagreeable odor of sweat. It may be followed by intertrigo and eczematous changes. The parts should be washed morning and evening with:

> ℞ Acid. tannic. ....gr. vi.
> Spirit. vin. dilut............................... ... ℥ iij.

and then dusted with:

℞ Acid. salicylic.,
  Flor. zinci. ....āā ℥ v.
  Talci præparat. .... ℥ x.

M. D. S. Dusting powder.

In hyperhidrosis of the palms of the hands, the parts are moist, often clammy and cyanotic.   The patients leave moist traces upon everything they touch.   If the hyperhidrosis is persistent, the epidermis, in places, is raised in vesicles, or it has a dull white color, and scales in more or less extensive shreds.   This is observed relatively often in chlorotic females who suffer from menstrual disturbances.   It often ceases spontaneously, but relapses may occur.   Hyperhidrosis of the feet is still more annoying to the patient.   Marked maceration and shedding of the epidermis produce violent pain and may render walking impossible. The disagreeable odor (bromhidrosis) is so pronounced that we can generally recognize the disease by the sense of smell.   Hebra showed that the bad odor results from the decomposition of the sweat which has been absorbed by the stockings.

In slight grades, daily foot baths, frequent change of stockings, and the daily application of the above-mentioned dusting powder, especially between the toes and stocking, will suffice.   In advanced cases, we may order unguent. diachylon Hebræ, which is smeared thickly on a piece of linen in which the foot is placed.   Small pieces are inserted between the toes.   The ointment is renewed every twelve hours.   In six to twelve days the macerated epidermis is cast off, and replaced by healthy epidermis.   The dusting powder should then be continued for a little while.

2. *Diminished Secretion of Sweat.   Anhidrosis (Hyphidrosis).*

Anhidrosis almost always occurs as a symptom of other diseases, such as diabetes mellitus, diabetes insipidus, chronic interstitial nephritis, cancer, cachexia in general.

Partial anhidrosis may also be secondary to other skin diseases, over the site of disease (eczema, prurigo, psoriasis, lichen, ichthyosis).

Finally, it may be the result of nervous influences.   According to Strauss, it occurs on the paralyzed side in peripheral facial paralysis, but not in central paralysis.   In paralyzed parts of the body, hyperhidrosis is sometimes noticed, sometimes anhidrosis, according to the occurrence of paralysis or irritation of the several nerve fibres which run along the peripheral nerves.

3. *Changes in the Quality of the Sweat.   Parhidrosis (Osmhidrosis, s. Bromhidrosis, Chromhidrosis, Hæmathidrosis, Urhidrosis).*

Changes in the quality of the sweat may affect its odor, color, or chemical constitution.

a. An unusual odor of the sweat is known as osmhidrosis or bromhidrosis.   In some cases, a bad odor is observed immediately after the secretion of the sweat; in others, it is the result of decomposition of the secretion.   Frigerio recently reported two cases in which the sweat of idiots smelled like musk.   Szokalski healed a nervous lady with hydropathic packs, and for two weeks the perspiration had the odor of violets. An urinous odor of the sweat may be detected in uræmic cases.

b. Abnormal color of the perspiration is known as chromhidrosis. Yellow, blue, green, black, and bloody sweat has been observed.

In jaundice the underclothing is not infrequently stained yellow by the perspiration.

A number of cases of blue perspiration (cyanhidrosis) have been reported. In 1869 Foote collected thirty-eight cases. It generally occurred in women, who were often anæmic and suffered from uterine diseases. The abnormal production of sweat was localized, most frequently on the lower lids, most rarely on the back of the hand, never upon the posterior surface of the body. Ipavic found shapeless masses of pigment in the secretion. The blue pigment has been found in a number of cases to be allied to indican. In Bergmann's case the blue color was produced by fungi.

Bloody sweat (hæmathidrosis) does not belong properly in this category. It is the result of unusual fragility of the finer cutaneous vessels, the blood being extravasated into the immediate vicinity of the sudoriparous glands or into their lumen. This condition is often simulated.

c. Among the anomalies in the chemical constitution of the sweat urhidrosis may be briefly mentioned. In this condition urea is excreted upon the integument in the form of white scales. This has been observed in suppression of urine in cholera or nephritis, and is often associated with uræmia (vide Vol. II., page 261).

B. Anomalies in the Secretion of the Sebaceous Glands.

1. *Increased Sebaceous Secretion. Seborrhœa.*

Two forms of this condition are distinguished, viz., seborrhœa oleosa and seborrhœa sicca. In the former, masses of fat are deposited on the surface of the skin, and either impart to it an unusually fatty gloss, or remain as thick, fatty scales and crusts. In seborrhœa sicca, there is excessive desquamation of thin scales and epidermis, mixed with drops of fat. Both forms may occur in the same individual. The disease may be general or local.

a. Seborrhœa of the scalp is most frequent during the first year of life. The scalp is covered more or less extensively with fatty scales or with yellowish-gray, greenish-gray, or blackish crusts, which may attain a thickness of several millimetres. The dark color is the result of admixture with dirt. These deposits never extend beyond the scalp. They often have a rancid odor, are peculiarly fatty to the feel, and are composed of drops of fat mixed with epidermis cells. If carefully removed, the underlying skin is found to be pale and unchanged. If the masses of sebum decompose and become irritating, the underlying skin reddens or it becomes eczematous and moist. The hairs are apt to come out while removing the masses of sebum.

If the condition is left untreated, the profuse secretion of sebum ceases in the second or third year, the accumulated masses of fat become dryer and more brittle, and are cast off by the growing hairs. But the condition should be relieved as quickly as possible. If the deposits are thin, the affected parts should be rubbed vigorously every morning and evening with ol. olivæ, ol. amygdal., or other fats, and the scalp then washed thoroughly with green soap. If there is a thick accumulation of fatty masses, they should be rubbed with oil every two hours, and a flannel cap placed on the head; when the crusts have been re-

moved, at the end of twelve to twenty-four hours, the parts should again be washed with soap. To prevent a relapse, the washing and inunctions should be continued morning and evening for some time.

In adults, the disease is rarely so severe as in infants. It occurs much more frequently as seborrhœa sicca. There is profuse desquamation of white, fat-containing epidermis scales, which make the hair appear unclean, and often covers the coat collar. This condition is known as pityriasis capillitii. It often gives rise to defluvium capillorum, and finally to alopecia. After recovery from the disease, the hairs are generally restored.

In some cases, no cause can be ascertained; in others, the condition is preceded by inflammatory diseases of the skin (eczema, erysipelas, small-pox); in still others by constitutional diseases (chlorosis, syphilis).

The treatment includes general and local measures. Iron, iodine, and mercury must be employed according to the causal indication. At night, the scalp should be rubbed with alcohol, in the morning with oil.

b. Seborrhœa of the face is most marked on the forehead, temples, nose, and chin. The parts have an unusually fatty gloss, the skin feels fatty; in rare cases, the fat collects in scales and crusts. The mouths of the sebaceous glands are unusually wide, and contain a more or less projecting mass of fat. Dust is apt to settle upon them, giving the patients a speckled, dirty appearance.

In seborrhœa of the nose, the cutaneous vessels situated between the openings of the sebaceous ducts are injected and sinuous. The condition is not infrequently present only at the period of puberty, and is especially severe in brunettes. Treatment is similar to that of seborrhœa of the scalp.

c. Seborrhœa of the genitals is especially apt to develop in males who suffer from phimosis. The smegma collects at first in the coronary sulcus, but may finally encrust the entire glans. In hot weather or after exercise, the secretion is apt to decompose, produces balanitis and balano-posthitis, and may finally give rise to excoriations and condylomata. The patients often suffer from herpes progenitalis, and, in children, the violent itching may result in onanism. If the smegma stagnates for a long time, preputial calculi may form.

The treatment consists of relief of the phimosis by gradual distention or operation, removal of the smegma by oily inunctions, and prevention of its reaccumulation by careful washing with soap, and introduction of charpie smeared with zinc ointment or with tannin and glycerin into the preputial sac.

Seborrhœa genitalium is also observed in females, especially in little girls. The sebum accumulates in the vicinity of the clitoris and between the labia majora and minora.

d. General seborrhœa occurs in adults as the result of chronic wasting diseases. The entire integument is covered with fine, fat-containing scales. This condition is termed pityriasis. The patients should be nourished as generously as possible, and should take lukewarm baths followed by fatty inunctions.

In the new-born, the production of sebum, which forms the vernix caseosa, sometimes continues for a few days. The little ones look as if covered by a thin, shiny membrane, which contains numerous fissures (ichthyosis sebacea). This may be associated with refusal to take nourishment, and unusually low bodily temperature, so that the children die in collapse. If they refuse to nurse, they should be fed with the

spoon, warming flasks applied to the body, a protracted warm bath (34° R.) given three or four times a day, and the entire integument rubbed with oil.

2. *Diminution of the Sebaceous Secretion. Asteatosis.*

(*Oligosteatosis.*)

Diminution of the sebaceous secretion may be congenital or acquired. The skin is unusually dry, has a tendency to form painful and often bleeding rhagades, desquamates more or less actively, and has a rough appearance.

This condition may be congenital, either alone or in combination with congenital diseases of the skin, for example, prurigo and ichthyosis. The acquired form may also be associated with certain skin diseases, such as psoriasis, lichen ruber, etc. It may be confined to the back of the hands and forearms in individuals who wash themselves frequently, especially if the water contains a good deal of lime or irritating soap. This is noticed particularly in the winter. The treatment consists of inunctions of vaseline or cold cream. Glycerin, which is such a favorite remedy among the laity, not infrequently aggravates the disease by extracting water from the tissues. The patients should wash less frequently and only in lukewarm water, and should wear gloves in winter.

3. *Anomalies in the Excretion of Sebum. Parasteatosis.*

a. Comedo.

I. SYMPTOMS.—Comedos are little black specks which correspond to the excretory ducts of the sebaceous glands, and in places are slightly elevated above the level of the skin. On pressure, the black speck is extruded, and is often followed by a yellow, spiral, thread-like structure. The latter consists of sebum, and, under the microscope, is found to be composed of fat and fatty epidermis cells, occasionally of cholestearin crystals. In many cases we also find downy hairs and acarus folliculorum.

Comedos develop most abundantly in those parts which are rich in sebaceous glands (forehead, nose, lips, chest, and back). They may occur alone, or are associated with seborrhœa. They are sometimes so numerous that the skin presents a black-speckled appearance, or in places they are so closely aggregated as to form wart-like prominences. They may also give rise to inflammation of the sebaceous follicles (acne).

Kuestner calls attention to the constant presence of comedos on the tip of the nose in the new-born.

II. ETIOLOGY.—The causes of the retention of the sebum cannot always be ascertained. In some cases it is the result of mechanical occlusion of the excretory duct, as is observed in workers in tar or petroleum, or in individuals who, despite the fact that their integument is fatty, rarely wash the face with soap. In others, it is the result of the production of unusually stiff sebum. Comedos often develop in anæmic and exhausted individuals, very often at the period of puberty.

III. TREATMENT.—The treatment consists of mechanical removal of the comedo by means of a watch key, etc. To increase the tonus of the dilated follicles, we may apply irritating washes, for example, Hebra's

green soap (vide page 336). This should be done at night. General treatment must be adopted in chlorotic, scrofulous, or anæmic individuals.

b. Milium.

1. Milium forms light-yellow or grayish-white papules, varying from the size of a millet seed to that of a pin's head, and which often are slightly elevated above the surface of the skin. Upon slitting the epidermis and exercising pressure, a grayish-white papule appears. Under the microscope this is found to be composed of lamellæ of epidermis cells and a fat-containing nucleus. This is the result of obliteration of the excretory duct of a sebaceous gland, so that an accumulation of epidermis cells takes place in a lobule, or in the entire gland, and dilates it. The epidermis passes over the papule, so that the former must be destroyed before the contents of the latter are laid bare. Milium is distinguished from comedos by the fact that the latter occupy the excretory duct of the sebaceous glands.

Calcification of milium has been observed in some cases. Wagner reported a case of colloid milium, i. e., the papules were the result of colloid degeneration of the epidermis cells.

The disease is most frequent upon the integument of the eyelids and adjacent parts of the cheeks and forehead, next upon the lips, the penis, and inner surface of the labia minora. They are sometimes so numerous upon the corona of the glans penis as to form a wreath.

2. The disease is often noticed at the periphery of cutaneous cicatrices, for example, those produced by burns, syphilis, or lupus. It is also frequent in parts which have been the site of previous skin diseases, for example, pemphigus. In some cases it seems to be the result of a primary anomaly of secretion, inasmuch as epidermis cells are deposited in abundance within the lobules of the gland, but do not undergo fatty degeneration and remain within the lobule.

Kuestner observed an abundant development of milium in children born prematurely.

If they are very numerous in the face, the epidermis should be nicked with a fine bistoury, and the contents removed by pressure.

PART III.

HYPERTROPHY OF THE SKIN.

A. HYPERTROPHY OF THE CUTANEOUS PIGMENT.

1. Nœvus.

Nævi are congenital accumulations of pigment in the skin. They form brownish or brownish-black, flat, and smooth patches (nævi spili), or they are uneven and warty (nævi verrucosi), or pedunculated (nævi mollusciformes), or covered with bristling, thick dark hairs (nævi pilosi). The size and number of the nævi are extremely variable. They are sometimes so numerous that the skin assumes a speckled appearance. They are situated not infrequently along the course of certain nerves, and, like herpes zoster, only upon one side of the body (nerve nævi).

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The formation of the pigment has been regarded, accordingly, as the result of trophic disturbances, and this view seems to be favored by the fact that other unmistakable nervous symptoms are sometimes noticeable. Nævi generally present a tendency to further growth. In later years they are occasionally the site of development of sarcoma or cancer, and may be the source of secondary infection of other organs. Anatomical examinations have shown that there is not alone an accumulation of pigment in the lowermost cell layer of the rete Malpighii, but that there is sometimes an abnormal development of pigment in the cutis and its obliterated vessels. The question of removal by operation comes up only with regard to smaller nævi.

2. Chloasma.

Chloasma is the term applied to an acquired development of pigment in the skin, and is either a primary (idiopathic) or symptomatic skin disease.

Idiopathic chloasma includes freckles (ephelides, lentigines). These form light-brown patches which may attain the size of a lentil, and are especially numerous in the face (nose and adjacent parts of the cheeks). But since they are also found on parts which are constantly covered with clothing, for example, the flexor surfaces of the arms and the genitalia, it is evident that the action of the rays of the sun is not their sole cause. Individuals with a delicate complexion are especially apt to be affected with freckles. They are most marked in the spring and summer, and grow pale or even disappear in winter. They are rare before the age of six years or beyond the fortieth year. They may be removed temporarily by remedies which produce an abundant desquamation of epidermis : soap inunctions, tincture of iodine, corrosive sublimate (gr. viij. : ℥ ij.). In many individuals, they disappear spontaneously at the completion of puberty.

The term traumatic chloasma is applied to those pigmentations which follow mechanical irritation of the skin. It is often observed in places upon which pressure has been exercised for a long time by bandages. etc., also after violent and repeated scratching (prurigo, eczema, scabies, pediculi). As the effect of clothes lice, tramps sometimes acquire such a dark color of the skin as to look like mulattoes (melasmas, melanoderma, nigrities).

Chloasma caloricum is the term applied to the dark color of the skin produced in many individuals (often within a few hours) by exposure to the sun and air.

Toxic chloasma may be produced by the application of irritating substances to the skin. The application of a mustard plaster or fly blister very often leaves a brown discoloration of the integument.

Among the symptomatic forms of the affection, chloasma uterinum deserves special mention. It forms brownish-yellow patches which, unlike the somewhat similar patches of pityriasis versicolor, are not shining, do not desquamate, and do not contain the fungus (mikrosporon furfur) peculiar to pityriasis. It is observed in women who menstruate irregularly, or suffer from diseases of the uterus and ovaries. In many women it occurs during pregnancy (chloasma gravidarum). It generally disappears at the menopause. The forehead, cheeks, lips, and chin are its most frequent sites.

Chloasma cachecticorum is observed, not infrequently, in marantic

individuals (malarial cachexia, marasmus of syphilis, cancer, pulmonary phthisis).

Pigmentations are not infrequently left over in places which have been the site of chronic skin diseases.

B. HYPERTROPHY OF THE EPIDERMIS. KERATOSIS.

Cutaneous changes which depend chiefly on hypertrophic conditions of the stratum corneum are known as keratoses. They may either exist alone or be associated with hypertrophy of the papillary body.

Among the keratoses which are confined to the stratum corneum, are a series of cutaneous changes which are so frequent that they merely require bare mention. These include callosities (tyloma), which are generally the result of frequently recurring pressure; corns, which are one variety of cutaneous callosity, and horns (cornua cutanea).

The keratoses, in which the papillary body is affected, include warts, condyloma acuminatum, and ichthyosis. The latter alone possesses medical interest.

Ichthyosis.

I. SYMPTOMS AND DIAGNOSIS.—Ichthyosis is a chronic, usually congenital disease, in which more or less thick deposits of epidermis form upon the skin.

In the mildest grades, the skin is covered by irregularly formed plates of epidermis, which are bounded by deep furrows. At the same time, the skin feels peculiarly rough and dry, and presents a great tendency to desquamation of the most superficial epidermis layers (ichthyosis simplex). If the central portions of the individual fields of epidermis are depressed, while the edges are slightly elevated, it is known as ichthyosis scutelata. In some cases the scales, particularly at the periphery, have a mother-of-pearl gloss. In many cases, they assume a greenish or greenish-gray color. The highest grade of development of the disease is reached in ichthyosis cornea and ichthyosis hystria. In the former, the scales of epidermis are as hard as horn, thick, curved; in the latter, they form prickle-like prolongations and prominences. As a matter of course, such changes produce great deformity, particularly since the nodules are not infrequently almost black.

Various forms of ichthyosis are sometimes observed in the same individual.

The condition may be general or circumscribed; the latter form is less frequent.

The first changes generally appear at the end of the first year of life, often not until much later. Cases of congenital ichthyosis have been reported, but good authorities maintain that these cases were really instances of seborrhœa. Hereditary influences are not exerted at once, but some time after birth.

The extensor surfaces of the elbows and knees are first affected, later the changes spread more and more, but the flexor surfaces of the joints, axillæ, genitals, palms of the hands, and soles of the feet almost always remain intact. In the face and scalp, as a rule, we find merely active desquamation; the hair of the scalp is unusually dry and has a tendency to fall out. The affected parts rarely perspire. Many patients complain of annoying pruritus. Deep, painful rhagades sometimes develop, but they rarely penetrate into the cutis. The thick, tense

epidermis may interfere with the free movement of the joints, so that they are kept almost constantly in a semi-flexed position. In marked ichthyosis of the face, impaired mobility of the lids and ectropium have been observed.

Ichthyosis is sometimes associated with other skin diseases (eczema, lupus, measles, small-pox).

Mapother has observed cardiac hypertrophy, which he attributes to disturbance of the cutaneous circulation. Increase and diminution of diuresis have been observed in this disease. In one case, W. Boeck described the development of a cataract and retinitis pigmentosa, with atrophy of the optic nerve.

II. ETIOLOGY.—Little is known concerning the causes of the disease. In some families it is hereditary, and Nayler was able to trace it back for six generations.

Sometimes only one child is attacked; in other cases, the male or the female children. The disease may also skip a generation.

According to Gaskoni, ichthyosis often appears in families which suffer from eczema, asthma, phthisis, and gout.

It is sometimes acquired, particularly in parts of the body which have been the site of chronic skin diseases.

Ichthyosis runs a chronic course, and generally lasts for life. Spontaneous recovery is exceptional. Exacerbations and remissions are observed not infrequently.

III. ANATOMICAL CHANGES.—The anatomical changes affect not alone the epidermis, but also the cutis and subcutaneous cellular tissue. The panniculus adiposus is considerably atrophied, and sometimes disappears almost entirely. In the cutis, we find hypertrophy of the papillæ, often to a very marked extent. Their vessels are unusually wide, sometimes thickened. The epidermis shows considerable increase of the cornified cell-layers, while the juicy cells of the rete Malpighii are sometimes reduced to a single layer. There is sometimes increase of the nuclei in the epithelium cells of the mucous layer. Some writers mention very distinct development of the prickle layer and an excessive amount of the intercellular substance. Brown and black pigment granules are found within and without the epidermis cells. J. Neumann observed atrophy of the sebaceous and sweat-glands.

IV. PROGNOSIS.—The prognosis is unfavorable, since the disease can only be relieved temporarily. However, there is no danger to life.

V. TREATMENT.—Active desquamation of the epidermis should be stimulated by inunctions of green soap at night, and a warm bath (30° R.), containing ℥ viij.–xvi. of soda, every day. This object may also be secured by protracted baths, wearing rubber garments, inunctions of cod-liver oil or resorcin ointment (3–20 per cent), etc. Internal treatment is useless.

C. HYPERTROPHY OF THE HAIRS.

Hirsuties.

(*Hypertrichosis.   Polytrichia.   Trichauxis.*)

In this condition, the hairs develop in abnormal numbers or in abnormal parts of the body. The condition may be congenital or acquired. The following are the principal forms:

a. Children are sometimes born with very long and abundant hair on the scalp, but this almost always drops off after a certain length of time.

b. In some cases, the entire body, including the face, is thickly covered with hair. This form is generally hereditary. The development of the teeth in such individuals is usually defective.

c. Thick hairs develop not infrequently upon nævi.

d. A beard sometimes appears in women, especially in those who are sterile or suffer from menstrual anomalies.

e. An abnormal formation of hair sometimes takes place on wounds and inflammations of the skin; also upon paralyzed limbs.

f. Some individuals present an unusual development of the hair of the beard and scalp, so that the hairs may flow to the ground.

If the abnormal growth is scanty, the hairs may be removed with tweezers, but permanent recovery is only effected by destruction of the hair-follicle. This may be done with caustic pastes and the galvano-cautery.

D. HYPERTROPHY OF THE NAILS.

Onychauxis.

The changes produced by hypertrophy of the nail substance belong to the domain of surgery. The nail may increase in thickness, or it becomes unusually long and broad, or both conditions are combined. It is sometimes bent like a horn or claw (onychogryphosis), or is raised from the nail-bed, or gives rise to inflammation of the nail-furrow (paronychia). Hypertrophic nails are usually rough, brittle, uneven, and discolored.

This condition may result from local injuries (pressure), or other chronic skin diseases (psoriasis, prurigo, lichen, ichthyosis, etc.) or constitutional diseases (chlorosis, syphilis, pulmonary phthisis).

E. CONNECTIVE TISSUE HYPERTROPHY.

Sclerema of Adults.

(*Scleroma. Sclerodermia.*)

I. SYMPTOMS.—Sclerema appears generally as a chronic change in the skin, which gives rise to a peculiar condensation, hardening, and retraction of the affected parts.

The changes sometimes occur in patches (partial) or they may be diffuse. They are found most frequently in the upper half of the body. They may develop imperceptibly so that, at the most, the patient's attention is attracted by a feeling of unusual tension and slight itching, rarely by rheumatoid pains, or they are noticed accidentally; at first the skin is sometimes œdematous, or there may have been preceding erysipelatous changes.

In the beginning, the skin is slightly elevated and contains a doughy infiltration. A striking condensation soon sets in and no longer permits the skin to be raised into folds. This increases more and more, and the skin becomes immovably adherent to the underlying tissues, the periosteum, sheaths of the tendons, or fasciæ of the muscles. The skin may look normal, or it is unusually pale and alabaster colored, or rosy-red to brownish-red. It sometimes contains more or less numerous and large

patches of pigment, in other cases certain regions contain an unusually small amount of pigment. The periphery of the sclerotic patches is sometimes very much congested, particularly when the process is advancing peripherally.

The longer the disease lasts and the more it advances the more marked become the symptoms in the parts first attacked. The originally elevated portions of the skin become more and more depressed, and form, in a measure, firm, band-like stripes which compress the underlying structures. Thus, the chest is sometimes found divided into two parts, or compression of veins produces œdema, etc. The skin may also grow as thin as paper, very red, desquamating, and exquisitely atrophic.

The cutaneous functions may remain entirely intact. The production of sebum and perspiration is usually unchanged. Cutaneous sensibility is generally retained, exceptionally it is blunted. The temperature of the skin may be increased, diminished, or normal. Other exanthemata (acne, herpes zoster, variola) develop not infrequently upon the sclerotic parts. Ulceration and gangrene of the skin have also been observed.

As a matter of course, the firm sclerotic skin, acting like a narrow, unyielding cuirass, will produce various functional disturbances and pressure effects. In diffuse facial sclerema, the face becomes rigid, destitute of folds, incapable of expression, and the patient lives, in a measure, behind a mask. Impaired mobility and ectropium of the lids set in, painful rhagades form at the mouth, which grows smaller and smaller. Paulicki described a case in which he was compelled to remove several teeth from the upper jaw in order to render possible the introduction of food. The condition grows still more distressing if the mobility of the arms has been almost abolished as the result of sclerotic changes around the elbows, hands, and fingers.

Diffuse sclerema in the neck interferes with the movements of the head. Sclerema around the joints of the limbs gives rise to stiffness of the joints, painful rhagades, and changes in position. In extensive sclerema of the integument of the thorax the patients complain of a feeling of constriction and anxiety. Sclerema of the penis may render erection impossible.

Similar changes are sometimes observed upon the mucous membranes (tongue, fauces, larynx, vagina).

As a rule, the disease is chronic, more rarely it runs an acute course. In the acute cases, the cutaneous changes are generally preceded by œdema of the skin. The sclerotic parts often become soft, while others are being diseased. Death occurs generally with the signs of increasing marasmus. Rossbach described a combination of sclerema and Addison's disease.

II. ANATOMICAL CHANGES.—The anatomical changes appear principally in the cutis and subcutaneous cellular tissue. The epidermis is almost entirely unchanged, with the exception of circumscribed proliferations of the rete Malpighii and accumulations of pigment. The cutis shows proliferation and condensation of the connective-tissue elements and elastic fibres. The vessels are narrowed in places. Upon their outer wall, and also in other parts, are observed nest-like accumulations of round cells. There is also an increase of connective tissue in the subcutaneous cellular tissue, while the adipose tissue proper disappears more and more. Accumulations of round cells are also found in this locality.

All other cutaneous changes appear to be secondary; they include accumulation of pigment in the cutis, dilatation of the excretory ducts of the sweat glands, hypertrophy of the smooth muscular fibres, etc.

The following lesions have been found in other organs : tubercular changes in the lungs and other viscera, cirrhosis of the liver and kidneys, cardiac hypertrophy, obliteration of the thoracic duct.

III. ETIOLOGY.—Little is known concerning the causes of the disease. Among those mentioned are erysipelas, injury, mental excitement, and cold. The disease is much more frequent in women than in men. It is exceptional in childhood, and generally develops beyond the age of twenty-five years. Cruse observed one case in which the disease developed a few days after birth.

We know as little concerning the nature of the disease as we do concerning its causes. Some authors regard it as an inflammation, others as a disease of the lymphatics ; the majority of writers regard it as a trophoneurosis. Eulenburg recently called attention to the intimate relations between scleroderma and facial hemiatrophy.

IV. PROGNOSIS.—The prognosis is not very favorable, although a few cases of recovery have been reported. The disease generally advances uninterruptedly, and proves fatal by exhaustion at the end of months or years.

V. TREATMENT.—The best results are obtained by massage, oily inunctions, and the application of emplast. mercuriale. Buelau recently cured a case with sodium salicylate (ʒ i. daily), which he administered for months. Good effects have been obtained from galvanization of the sympathetic and of the affected parts of the skin.

APPENDIX.

Sclerema Neonatorum.

I. ETIOLOGY.—The disease is similar to scleroderma in certain external appearances, but, anatomically and etiologically, it is entirely different. It generally attacks children during the first few months of life ; in rare cases, it begins in the second, or even in the third year ; in a few instances it was congenital. It occurs most frequently in the winter, in the children of the poorer classes. The children were not infrequently born prematurely, or were asphyxiated at birth, and suffered from atelectasis, bronchitis, broncho-pneumonia, gastro-enteritis, or congenital valvular disease.

II. SYMPTOMS.—The first changes appear upon the integument of the calves ; then it extends to the feet, thighs, abdomen, chest, face, and upper extremities. The skin is at first œdematous and slightly reddened ; then it becomes peculiarly rigid, cannot be raised in folds, and is very cold. It is peculiarly smooth, and either slightly reddened or assumes a waxy pallor. Cutaneous sensibility is diminished or abolished. The more the cutaneous changes extend the more the movements of the child are interfered with. If the face is attacked, it acquires a rigid, unchangeable expression, like that of an old man. The mouth can hardly be opened, and infants are unable to nurse. The integument is as cold as that of a frozen corpse. The bodily temperature is constantly lowered (22° C.). The pulse becomes slow and feeble. The second heart sound is hardly audible. The children are generally apathetic, occasionally they moan feebly.

Cyanosis and jaundice are observed not infrequently as the result of changes in other organs.

Death generally occurs after increasing collapse and progressive diminution of temperature. More rarely the hardened portions of skin soften and recovery ensues, after gradual elevation of the bodily temperature. Improvement is sometimes only temporary. Death may occur in a few hours ; the disease rarely lasts longer than two weeks.

III. ANATOMICAL CHANGES.—The anatomical changes consist mainly of rigidity of the subcutaneous cellular tissue. Demme found fatty degeneration of the heart muscle ; Parrot noticed ecchymoses in the mucous membrane of the bladder. Langer has shown that, in the new-born, the fat contains larger amounts of the solid fatty acids (palmitin and stearin acids) than in adults, and is therefore more solid, and melts at a higher temperature. Hence a slight diminution of the bodily temperature will make it firm and rigid. Such a depression of temperature may occur very readily from disturbances in the cutaneous circulation, which are apt to develop in the diseases mentioned under the head of Etiology.

IV. PROGNOSIS AND TREATMENT.—The prognosis is almost always fatal. Repeated warm baths (half-hour) at 37° C. should be ordered. After the bath, the child is placed in a warm bed, and warming flasks applied to the body. Artificial feeding must be resorted to. Camphor may be injected subcutaneously. Careful massage and faradization of the muscles have also been employed.

PART IV.

ATROPHY OF THE SKIN.

A. ATROPHY OF THE CUTANEOUS PIGMENT.

Leukoderma.

(Leukopathia. Achromatia.)

A deficiency of cutaneous pigment is shown by the unusually white color of the skin, either in patches or diffusely. Congenital conditions are known as albinism, acquired conditions as vitiligo.

All forms of pigment atrophy are more frequent in negroes.

a. General albinism is shown by the brilliant white color of the skin. The hairs are yellowish-white, and have a silky gloss. The iris and choroid are poor in pigment, and present a red reflex in direct sunlight. Heredity is observed in some cases.

b. Partial albinism consists of the formation of patches of light skin. They are most frequent upon the genitalia, scalp, chest, back of the hands and fingers, and are not infrequently situated on symmetrical parts. The hair on the affected parts is also poor in pigment, and has a gray or white color. The patches generally remain stationary, but sometimes they increase gradually in size.

c. Vitiligo occurs, in the majority of cases, as an idiopathic change in the integument. Violent excitement, exhausting diseases (typhoid fever), and injury are sometimes mentioned as causes. The symptoms often appear quite suddenly. Naecke states, with regard to his own

case, that he noticed the first changes during a railway trip. As a rule, the disease develops in adults, but cases have been reported at the age of twenty-one days, five and eighteen years respectively.

This condition is easily recognized. The skin contains light white patches with very darkly pigmented borders. In one of my cases, a small, dark pigmented spot was found near the middle of each white patch. The patches gradually increase in size, coalesce, and finally occupy the greater part of the body. At certain times the patches may increase rapidly in size and number; then, again, they may remain stationary for a long time. They often follow the distribution of the cutaneous nerves. The functions of the skin are unchanged.

Vitiligo occurs symptomatically in those parts of the skin which have been subjected to injury and ulceration (white color of cutaneous cicatrices). It develops not infrequently in localities which have been subject to protracted pressure by bandages, etc. It may follow various cutaneous eruptions.

These conditions are characterized anatomically by deficiency of pigment in the lowermost layers of the rete cells. Treatment is powerless.

B. Atrophic Changes in the Hairs.

1. *Canities.*

(*Poliosis.*)

Gray hair may be congenital or acquired. It is congenital in albinism; in diffuse albinism it affects all the hairs on the body, in partial albinism only those places which are destitute of pigment. In some individuals, small patches of gray hair appear congenitally, independently of albinism.

Senile canities is one of the physiological forms of this condition. It begins, as a rule, in the temples, and then extends to the hair of the scalp and beard. In some families it appears very early. The process is the result of an insufficient supply of pigment by the hair papilla, so that the cortical layers of the hair become poor in pigment. The gray color begins at the hair bulb. At first, the disturbances in the formation of pigment are occasionally temporary, so that the hairs present alternate gray and dark portions. If the hair grows gray independently of age, the condition is known as premature canities. This is observed after typhoid fever, syphilis, erysipelas, etc., if the hairs have fallen out and then grown again. They not infrequently become gray and scanty. The hairs may also grow gray after skin eruptions and cicatrices, and in vitiligo. It is well known that premature grayness may be the result of loose habits and violent emotions. It may also be the result of nervous influences. Thus the hairs sometimes grow gray in hemicrania and neuralgia in the affected nerve tracts. Berger observed it in facial paralysis.

The hairs may grow suddenly gray in consequence of violent emotions. In one of these cases, Landois and Lohmer observed air-bubbles in the gray hairs, but Kaposi claims that they may also be present in hairs which are not gray.

2. *Alopecia and Atrichia.*

1. Complete absence of hair may be congenital or acquired. Congenital atrichia is often associated with absence of the teeth; sometimes the hairs developed at the age of two or three years. Acquired atrichia occurs occasionally after violent emotions. Todd described a case in which a man who was struck by lightning lost all his hair and nails; in another patient the loss of hair followed concussion of the brain. Crisp observed atrichia after malaria, which was associated with anæsthesia of the left side. It is evident, therefore, that this condition is influenced by nervous disturbances. In some cases, however, no cause can be discovered. This happened in two of my own cases. The falling out of the hair (defluvium capillorum) generally begins on the scalp, then extends to the beard, eyebrows, eyelids, axillæ, and mons veneris, so that the patients are entirely destitute of hair. This sometimes takes place within a few weeks.

2. Falling out of the hair is a physiological process in old age (alopecia senilis). It is generally preceded by canities. The senile defluvium either begins on the forehead and temples, and gradually extends to the vertex, or the first bald spots appear at the vertex. The bald spots are glossy and often covered with yellow or dirty gray scales of accumulated sebum. Senile defluvium is observed occasionally in the beard, but this is exceptional. The process must be attributed to senile changes in the skin. Koelliker lays stress on obliteration of the vessels in the hair papillæ.

3. Falling out of the hairs in earlier years is known as premature alopecia. In some families it is the result of an inherited predisposition; in other cases, of nervous influences (mental strain, worry, grief, hemicrania, dissipation).

Alopecia sometimes follows infectious and constitutional diseases (typhoid fever, febrile infectious diseases in general, pulmonary phthisis, cancer, and marantic conditions). Many women suffer from very marked alopecia after childbed. If the condition is only temporary, a new growth of hair makes its appearance after a while, and is occasionally even more abundant than the preceding growth.

Alopecia is sometimes the result of local changes in the scalp. It is associated not infrequently with seborrhœa of the scalp, also with erysipelas, eczema, prurigo, lupus, psoriasis, lichen, favus, herpes tonsurans, and with variola, sycosis, and syphilides of the scalp. It is incurable if these diseases have given rise to destruction of the hair papillæ and follicles.

4. The various forms of alopecia must be treated in almost the same way. At first, the causal conditions must be taken into consideration. Locally we should apply irritating alcoholic lotions, followed by inunctions with oils, for example :

R Spirit. vin. dilut.................................... ℥ iij.
  Acid. carbolic.................................... ℨ ss.

M. D. S. To be rubbed in morning and evening.

R Ol. macidis .... ℥ iij.
  Ol. oliv.................................... ℥ x.

M. D. S. To be used as hair oil; or,

R Acid. carbolic .... ℈ ss.
  Sodii salicylat .... ℈ iss.
  Vaselin .... ℥ x.

M. D. S. To be used as pomade.

3. *Alopecia areata s. Celsi.*

1. The changes in alopecia areata consist of the complete loss of hair in sharply defined, round spots, which look as if shaved with a razor. The spots gradually increase in extent. If a number of spots are present, adjacent ones often coalesce and produce irregularly-shaped patches. In very advanced cases, there may be almost complete loss of the hair on the head, and the beard may be similarly affected. The cutaneous sensibility of the bare spots is sometimes diminished. The hairs which are situated at the boundaries of the bald spots are often unusually loose. In some cases the disease is hereditary or it appears in several members of the same family. It lasts for months and years. There is often a sudden remission in the symptoms, followed by the growth of downy hairs, later of normal hairs.

2. The majority of writers attribute the disease to trophoneurotic disturbances. In our opinion some of the cases, at least, are parasitic in their origin. The distribution of the spots, their shape and growth, favor the latter view more than the former. The treatment is the same as that of alopecia.

4. *Trichorhexis.*

1. The ends of the hairs not infrequently split up. This occurs particularly in long hairs, most frequently in the long hairs of the head in women, more rarely in the long hairs of the male beard. It is probably a nutritive disturbance, perhaps the result of the excessive growth of the hair. The treatment consists in cutting the hair.

2. Trichorhexis nodosa occurs almost exclusively in the hairs of the beard, rarely in those of the eyebrows. In places the hairs form nodular distentions, which are sometimes so numerous as to present a rosary-like appearance. The hairs are situated firmly in their follicles, but readily break spontaneously or upon traction on the individual nodes. In places, they run into spherical prominences and present a gnawed appearance. Under the microscope it is found that the changes begin with a slight spindle-shaped dilatation of the hair. Next the cortical layers in this locality split up, while the medullary parts are often filled with large drops of fat, and press outwards more and more. But in many places the medullary cells are entirely absent. Finally, the cortical fibres of the upper and lower portions of the hair look like two brushes which are pushed into one another. The causes of the disease are unknown. Wolfberg believes that many cases are mechanical in their origin, and result from very strong friction of the hairs in drying them after washing. I recently cured two cases by advising the parties to discontinue rubbing the head after washing. Whether all cases of trichorhexis are due to this cause, is not settled.

C. Atrophy of the Cutis Tissue.

Xeroderma.

Kaposi distinguished two forms of this disease, one running its course with, the other without the formation of pigment in the skin. The skin appears peculiarly tense, thin, and shiny, or is covered with small scales ; its sensibility is retained, but it cannot be lifted readily into folds. The movements of the limbs are impeded, and the latter are kept semi-flexed; ectropium and narrowing of the nostrils and mouth become noticeable. In those forms which are associated with the formation of pigment, cancer and sarcoma of the skin developed in a number of cases. The prognosis is unfavorable.

PART V.

NEUROSES OF THE SKIN.

Itching of the Skin. Cutaneous Pruritus.

I. Etiology.—Cutaneous itching is an annoying symptom in many skin diseases, but we refer now only to that form which is independent of anatomical changes in the skin, and constitutes an independent functional disturbance of the cutaneous sensory nerves. It is observed not infrequently in old age, perhaps as the result of senile processes of involution in the skin. In other cases it is the result of general diseases and local affections of internal organs, such as diabetes mellitus, Bright's disease, jaundice, cancer, diseases of the liver, stomach, uterus, and ovaries. Pruritus is sometimes noticed at a very early stage of cancer. Some women complain of intolerable itching during pregnancy, and, according to Head, this symptom gives rise not infrequently to premature labor if it appears in the second half of pregnancy. Duhring applied the term pruritus hiemalis to those cases which occur in certain individuals on cold autumn and winter days. Pruritus has also been observed after violent excitement.

II. Symptoms.—The itching is sometimes felt constantly, sometimes it occurs in paroxysms. In the latter event, it appears particularly at night, or when the patient is very warmly clad. Mental emotion and the dread of an attack may also provoke a seizure. The patients often fall asleep quietly, but are roused during the night by the itching. In many the itching is so violent that they are compelled to avoid society, since they are unable to resist the desire to scratch. The patients are often pale, emaciated, and excited; they may even be driven to insanity and suicide.

The scratching leaves traces behind, in the shape of wheals, nodules, eczema, pustules, and excoriations. It is often difficult to decide whether the eruption or the pruritus is primary.

III. Prognosis.—The prognosis is favorable only when the causes can be removed. It is unfavorable in the pruritus of old age, Bright's disease, etc.

IV. Treatment.—The etiological indications must first be met. In addition, the skin should be anointed morning and evening with carbol-vaseline (3 : 50), and the following prescription ordered: ℞ Potass.

bromid., ℥ iij.; Ext. belladonnæ, gr. ivss.; Acid. carbolic., gr. xv.; Pulv. et succ. liq., q. s. ut ft. pil. No. 50. D. S. Two pills to be taken four times a day.

A large number of other remedies have been employed in this disease. We may mention the following; Veratrine, chloral hydrate, morphine, etc., douches or baths with soda, corrosive sublimate, etc., applications of ether, alcohol, chloroform.

PART VI.

PARASITES OF THE SKIN. DERMATOSES PARASITARIÆ.

A. ANIMAL PARASITES. DERMATOZOONOSES.

1. *Itch.* *Scabies.*

I. ETIOLOGY AND ANATOMICAL CHANGES.—The term itch is applied to an artificial eczema which is partly produced by the direct irritation of the acarus scabiei (sarcoptes hominis), partly by the scratching superinduced by the intolerable pruritus occasioned by the parasite.

The acarus scabiei belongs to the family of acarinæ and to the class arachnidæ. Each patient harbors many more female than male mites.

The female is larger than the male (the former is 0.27 to 0.45 mm. long, 0.2 to 0.35 mm. broad; the latter is 0.23 to 0.25 mm. long, 0.16 to 0.20 mm. broad), and, to the naked eye, appears as an irregularly round, gray, slightly transparent little nodule, which is barely visible. Under the microscope, it is found to be shaped somewhat like a crab, and pendulum-like movements of its eight feet and head are often observed. It becomes more transparent without undergoing any other changes, if a solution of potash (1 : 3) is added to the preparation.

The head is situated at the anterior extremity upon the dorsal surface (Fig. 139). It possesses two pairs of mandibles with three segments, and, to the outside of these, two three-jointed palpi provided with bristles. The lateral surfaces of the body present numerous constrictions, and from them project two anterior and two posterior pairs of feet. The anterior pairs have five segments , and at their free end carry a pedunculated sucking disk (ambulacra); the posterior pair possesses only three segments, and terminate in long bristles. Upon the broad dorsal surface are found numerous furrows and a number of rows of spines, the middle ones being convex anteriorly, the posterior ones convex posteriorly. In addition, there is a row of more or less pointed and long thorns. At the posterior end of the body, they are arranged in four rows, fourteen in number.

Upon the ventral surface of the female, the head can readily be traced from the œsophagus. Near the posterior extremity is a fissure which leads to the vagina; more or less developed ova are often visibile in the belly (Fig. 140). At the posterior extremity is found the anal opening, with long bristles on each side. No respiratory organs can be discovered, and the animals can live for a long time when deprived of air. The female acarus is said to live twenty to sixty days.

The male acarus is smaller than the female, and the posterior pair of feet possesses a pedunculated disk, while the third carries a long bristle, as in the female. There are a smaller number of thorns and spines on

the dorsal surface. Upon the ventral surface is seen the fork-shaped penis, situated in a horseshoe-like sheath of chitin (Fig. 141).

Probably a single act of sexual intercourse suffices for the production of a large number of viable ova. The latter are said to number about fifty, one or two ova being deposited on a single day. According to Gudden, the male dies six to eight days after intercourse.

When a female acarus is impregnated, it bores a passage (acarus burrow) through the stratum corneum into the rete Malpighii. This process can be traced by placing a living acarus on the skin. It bores

FIG. 139.
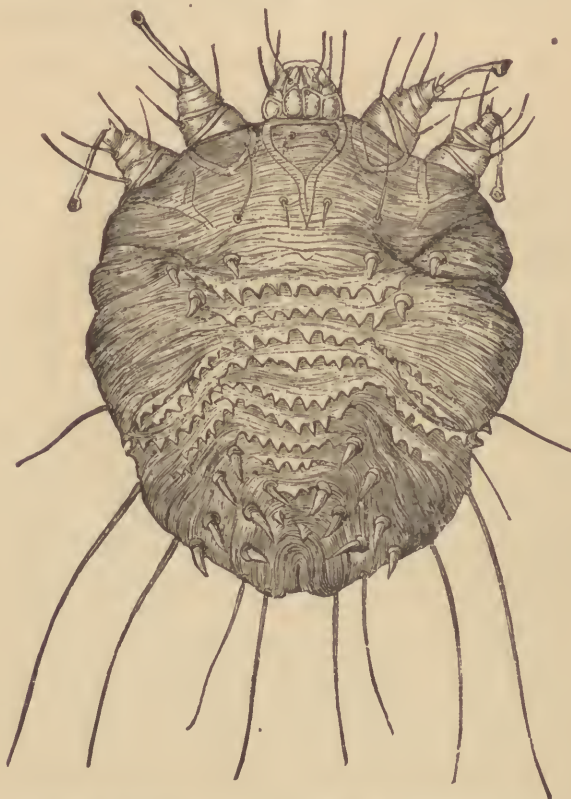
Female sexually mature acarus, seen from dorsal surface. Enlarged 300 times. After Kaposi.

its way to the deeper layers of the epidermis by aid of the mandibles. The burrow is generally curved or S-shaped; it often contains dirt or coloring matters so that it appears as a punctate, black streak. The animal is situated at the end of the burrow, and not infrequently can be recognized as a light spot with the naked eye, and can often be removed by the introduction of a needle. The length of the burrow is generally 0.5 to 1.0 cm., sometimes as many as 5 cm. The mite may bore a distance of 0.5 mm. in a day.

Under the microscope, it is found that the burrow begins at the

surface of the epidermis with a slight, funnel-shaped dilatation, and also ends in a slight enlargement. The female is always found at the blind extremity of the burrow. As it makes its way into the skin, it leaves ova in its track, the older ones being at the entrance, the youngest ones immediately behind the mother. The number of ova may amount to fifty, but on the average only ten to twenty are present. Mingled with the ova are black granules of fæces. The mite dies after having deposited a sufficient number of ova.

The acarus bores deeper and deeper into the skin, because it finds nourishment only in the juicy cells of the rete Malpighii, while the irri-

FIG. 140.
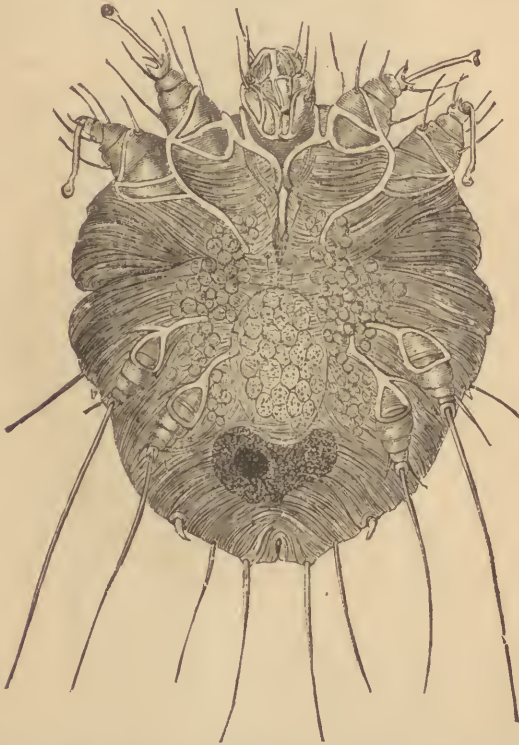
Female sexually mature acarus, seen from the ventral surface. Enlarged 300 times. After Kaposi.

tation which it exercises produces cornification of the surrounding epidermis cells, so that it is compelled constantly to seek new layers of epithelium. The irritation is also shown by inflammatory changes in the cutis below the burrow, so that papules, vesicles, and pustules form, and elevate individual portions of the canal.

The female acari alone are found in the burrow, the males are generally found in its vicinity in superficial furrows of the epidermis.

The ova are oval in shape, 0.16 mm. long and 0.11 mm. broad. The youngest present numerous fission processes, while the older ones contain the rudiments of the head and feet. In six to twelve days the ovum develops into a larva.

The larva (vide Figs. 143 and 144) breaks through the shell of the ovum, and makes its way through the burrow to the surface of the epidermis. According to some writers, it makes its way to the surface through air holes in the burrow. On the average, they attain a length of 0.15 mm. and a breadth of 0.10 mm. Several free larvæ are often found in the burrow. After they have reached the surface of the epidermis, they burrow a superficial nest in which they develop further.

The larvæ have only six legs, and present no sexual differences. They moult three times, according to some writers, four times. After the first moult the animal possesses eight legs, after the third moult it is sexually mature. According to Gudden, the first moult takes place from the fourteenth to seventeenth days, and each moult lasts five days.

The disease is always the result of the direct conveyance of the larvæ,

Fig. 141.

Male acarus seen from the ventral surface.   Enlarged 300 times.   After Kaposi.

but this requires prolonged and intimate contact with patients suffering from itch. It occurs most frequently from sleeping in the same bed, and is rarely the result of contact with the hands. If nurses and mothers suffer from itch, the nursling is often infected, because the breast is a favorite site of the mites. It has been erroneously held that the disease cannot be conveyed in the underclothing.

Scabies can be conveyed not alone from man to man, but also from animals to man. The parasite has been found in the dog, cat, horse, cow, sheep, rabbit, fox, elephant, etc.

Certain trades (shoemakers, smiths, bakers, tanners) present a predisposition to the disease, while others, especially cigarmakers, are generally not attacked.

The disease is generally more frequent in the winter, and is more common among men than among women.

II. Symptoms.—The favorite habitats of the itch-mite are those places in which the horny layer of the epidermis is thin, and the cells of the rete Malpighii are juicy. Such places are: the skin between the fingers, the flexor surfaces of the wrist and phalangeal joints, the palm of

Fig. 142.

Acarus burrow, cut from the integument of the loins. Slightly enlarged. After Kaposi.

the hand in children and individuals with a delicate integument, the extensor surface of the elbow, the anterior axillary fold, the nipple and umbilicus (particularly in women), the genitalia, the region of the trochanters, buttocks, knee, and inner border of the foot. The parasite is

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particularly abundant upon the buttocks in individuals who work in a sitting position, for example, shoemakers. The acarus is also apt to be found in parts which are subjected to prolonged pressure by bandages, etc. Hebra observed itch burrows upon the mucous membrane of the urethra. The face is generally unaffected, except in nurslings who acquire the disease from the mother or nurse.

The cutaneous changes produced by the parasites and by scratching are not always identical locally. The itching produced by the acarus is always radiated to parts which remain free from the parasite, and such parts are also scratched by the patients. The scratch efflorescences are found chiefly in parts which are easily reached by the patient's hand (anterior part of chest and abdomen, inner surface of thighs and knees).

Fig. 143.   Fig. 144.

Larva with six legs, seen from the ventral   Larva during second moult. After Kaposi.
surface.

The efflorescences of scabies are composed of papules, vesicles, pustules, crusts, and excoriations.

The term scabies norwegica is applied to very severe and inveterate cases, in which thick crusts form, particularly upon the face and scalp. The finger nails are also affected, being discolored, fissured, and thickened. After being treated with potash, the nails are found to contain innumerable ova, larvæ, and nests of acari (vide Fig. 145).

The sole complication is the enlargement of the lymphatic glands in the vicinity of extensive eczema. Suppuration of the buboes has been observed after extensive changes on the penis.

The patients generally complain of nothing beyond intolerable itching, particularly at night. This ceases during the course of febrile diseases, because the mature acari are killed by the increased bodily tem-

perature. When the temperature returns to the normal, the ova deposited in the burrows mature, and the symptoms then return.

III. DIAGNOSIS.—As a rule, the diagnosis is easy, but is only positive after the finding of the mites, ova, or larvæ. The burrows, which should be looked for between the fingers and upon the penis, are also characteristic, but they may be subjected to such severe scratching that it is hardly possible to find either them or the parasite. Even in such cases the diagnosis is usually easy, if we bear in mind the distribution of the efflorescences.

IV. PROGNOSIS.—The prognosis is good, since we are able to kill the parasites, and thus remove the cause of the cutaneous lesions.

V. TREATMENT.—In order to kill the parasites, the following should be rubbed into the skin morning and evening for two successive days:

R Balsam. Peruvian.,
Styracis liquid ....āā ʒ i.

M. D. S. To be rubbed in morning and evening, and after the sec-

FIG. 145.
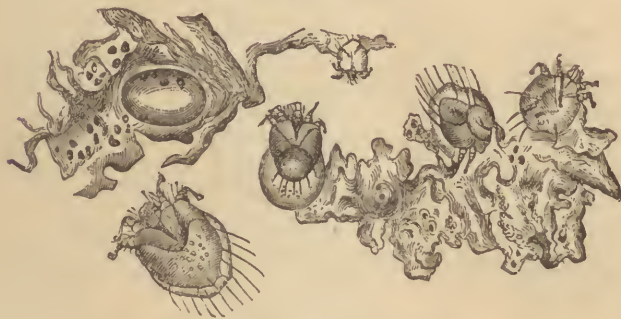
Crusts from scabies norwegica.

ond day a warm bath should be taken daily for three days and the parts washed with soap. In addition, the underclothing and bedding should be changed and boiled, the remainder of the clothing exposed to hot vapor in order to kill any parasites which may be present in these articles.

To relieve the eczema produced by the mites, we recommend oily inunctions, bland ointments, or the ordinary remedies in eczema.

We may mention some of the other remedies which have been employed in this disease: Carbolic acid, naphthol, naphtholin, ol. bergamottæ, sulphur, mercury, tar, etc. The following ointments may be mentioned:

R Calcis...................................... ʒ vij.
Sulphur. citrin............................... ʒ xiij.
Coque cum. Aq............................... ʒ xvi.
Ad remanent................................. ʒ x.

D. S. To be rubbed in.

R Flor. sulphur.,
Ol. cadini.................................āā ʒ v.
Sapon. virid.,
Axung. porci ....āā ʒ x.
M. D. S. To be rubbed in.

℞ Styrac. liquid,,
    Flor. sulphur.,
    Cretæ albæ.................................... ....āā ℈ iij.
    Sapon. virid.,
    Axung. porci............... ....āā ℥ vi.
    M. D. S. To be used externally.

If the ointments irritate the skin too strongly, they may give rise to albumi-
nuria, but this is a rare event.

2. *Acarus Folliculorum.*

(*Demodex folliculorum.   Macrogaster platypus.*)

In many individuals this parasite is found in the sebaceous and hair
follicles, but does not give rise to any other cutaneous changes.   It is
found with special frequency on the bald head of old people.

Fig. 146.

next upon the forehead, cheeks, nose, upper lip, and external
auditory canal.   It may be obtained by expressing the con-
tents of the follicles, and then placing the latter, with a
drop of oil, upon the object glass.   As many as twenty
have been found in a single follicle.

The anatomical (vide Fig. 146) characteristics are: An
elongated, cylindrical, worm-shaped animal, 0.08–0.12 mm.
long, 0.02 mm. broad; at the anterior end are two man-
dibles and two lateral palpi; on the thoracic portion, four
pairs of feet; the abdomen is four times as long as the
anterior portion.   Smaller, six-legged mites have also been
described.

When conveyed to animals, it is said that severe cutane-
ous lesions are produced.

Acarus folli-
culorum.
Enlarged.

3. *Lice.   Pediculi.*

There are three varieties of lice, viz.: Pediculus capitis,
pediculus vestimentorum, and pediculus pubis.

a. Pediculus capitis (head louse) is found only on the
scalp.   It is about two mm. long, and has six feet provided
with hooks.   The females are always more numerous than
the males.   In fertilization, the female sits upon the male.
The former lays about fifty ova, or nits.   These adhere to
the hairs by means of a sheath of chitin which surrounds the
hairs (Fig. 148, *a*).   The oldest ova are those situated next to the scalp.
At the end of three to eight days the young escape from the ova, and are
fully developed in eighteen to twenty days.   Within six weeks, the fe-
male may bear six thousand young.

An artificial eczema is produced upon the scalp, inasmuch as the
lice give rise to pruritus, and the consequent itching mechanically irri-
tates the scalp.   In combing, the painful spots are spared, so that the lice
find favorable conditions for development within the sticky hairs and
upon the bleeding skin, which is covered with pustules or crusts.   Finally,
the hairs form a densely matted mass, containing pus, crusts, and blood,
and which often has a nauseous and quite characteristic smell.   The ec-
zema often extends to the adjacent skin, the neighboring lymphatic
glands become swollen, the patients are sleepless on account of the in-

tolerable itching, lose their appetite, and run down in health. The back of the head is particularly apt to be affected.

In making a diagnosis, our attention should be directed to the presence of nits; the nearer they are to the tips of the hairs the older is the disease.

Treatment consists of the application to the hair of ung. hydrarg. cinereum, or of the following:

R Ol. petri italici .... ℥ iiss.
Ol. olivæ........ .... ℥ i.
Balsam. Peruvian................................ ℥ ij.
M. D. S. Externally.

FIG. 147.
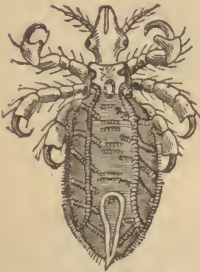
Male head louse. Enlarged.

FIG. 148.

FIG. 150.

FIG. 149.

Female clothes louse. Enlarged.

Hair with chitin sheaths and nits. Enlarged.

Crab louse. Enlarged.

In this manner the lice and nits are killed; they must then be removed by disentangling and combing the hair. The chitin sheaths adhere very firmly to the hair, and are removed more readily by washing with vinegar.

b. Pediculus vestimentorum (clothes louse) is the largest variety of louse in man, and attains a length of three to five millimetres (Fig. 149). Its habitat are the folds of clothes, particularly of the shirt. It is found chiefly on the neck and between the shoulder blades, on the sacrum, nates, external surface of the thigh, and immediately above the wrist-

joint. It deposits its ova in a rosary-like arrangement within the folds of the skin, and rarely leaves its habitat except to seek nourishment on the skin. It is, therefore, rarely found on the skin, but must be sought for in the folds of the underclothing. Its sting produces pruritus and wheals. Individuals who have suffered from body lice for a long time present open wounds, crusts, pustules, excoriations, even furuncles and extensive ulcers. When the efflorescences recover, they are followed at first by white cicatrices, but later they give rise to a deep-brown, even blackish diffuse pigmentation of the skin.

The treatment consists in placing the clothes in a vessel heated to 60–65° R., and thus killing the parasites. The eruption is treated in the ordinary way.

c. Pediculus pubis (crab louse) is only one millimetre in length (Fig. 150), and is found most frequently in the hair on the genitalia. It may also occur in the hairs of the axillæ, limbs, chest, beard, and eyebrows, but not upon the scalp. It is acquired most frequently during coitus, and gives rise to itching and eczema. Treatment similar to that of pediculus capitis.

4. *Fleas. Pulices.*

a. The common flea, pulex irritans, has its habitat in the folds of the underclothing. Its sting produces a minute extravasation of blood, which is surrounded by a hyperæmic zone, which grows pale on pressure. The latter also disappears spontaneously with great rapidity, while the petechia lasts for days. Dirty individuals are often covered with flea bites, and if they are dull and suffer from high fever, difficulty in diagnosis may arise if typhoid fever is prevalent. Chief importance must be attached to the distribution of the petechiæ, and to the presence of roseola around some of them. In individuals with a delicate skin, fleas give rise to wheals.

b. Sand flea (pulex penetrans) is found in America, most frequently on sandy beaches. The female bores into the skin, gorges itself with blood, and in two to five days produces inflammatory changes in the skin, which may terminate in ulceration, gangrene, and even erysipelas, lymphangioitis, and tetanus.

5. *Bed Bug. Cimex lectularius.*

The sting of the bed-bug produces wheals, not infrequently a reflex eruption of urticaria, which is attended with annoying pruritus. As a result of the scratching, the wheals are often excoriated and covered with crusts. The diagnosis is not always easy.

6. *Guinea Worm. Filaria Medinensis.*

This worm is found chiefly on the west coast of Africa. It may attain a length of one metre. Its habitat is the subcutaneous cellular tissue which it probably reaches from within, inasmuch as it is swallowed in the drinking-water and carried by the blood-vessels to the periphery of the body. It gives rise to abscesses of the skin, ulcerations, furuncles, and gangrene, produces fever, and even convulsions. If it projects

from an open wound, it should be wound around a little stick, and gradually withdrawn in the course of a few hours.

7. *Cysticerci of the Skin.   Cysticercus cellulosæ subcutaneus.*

Cysticerci of the skin generally form round or slightly flattened tumors, which are usually as large as a hazelnut.   They are painless on pressure, have a peculiar, firm, cartilaginous resistance, and are movable to a certain extent.   They are most apt to be mistaken for swollen lymphatic glands, and a positive diagnosis can only be made by extirpation.   We then find a dull white vesicle which, when cut open, discharges a clear fluid, and, upon its inner surface, contains the head which is recognizable externally by a thickening and slight depression of the vesicle.   The head often moves vigorously under the microscope (vide Vol. II., Fig. 29).

Hundreds of cysticerci are often found in one individual, in other cases only a few.   They may gradually grow smaller and disappear, while new parasites sprout up in other localities.   Cysticerci are often found in other organs, viz., the brain, eye, and other viscera.

In rare cases, the patients also harbor tapeworms.   Self-infection is conceivable only in the event that proglottides passed from the intestines into the stomach, and were there dissolved by the gastric juice.

The treatment consists of extirpation of the tumors, if they are not too numerous.

B. Vegetable Parasites of the Skin. Dermatomycoses.

1. *Pityriasis versicolor.*

I. Symptoms and Etiology.—Microsporon furfur, the fungus of pityriasis versicolor, proliferates in horny layers of the epidermis, and, according to Gudden, also penetrates the epidermoidal portions of the hair follicles.

The skin is covered with orange-yellow, brownish-yellow, or dark-brown patches which are slightly elevated above the adjacent skin, and glisten slightly or not at all; in old cases, they are scaly and fissured.   They can be readily removed by scratching with the finger-nail. This lays bare the reddened cutis which bleeds from numerous small openings.

If the epidermis scales are placed on an object glass, and a drop of

Fig. 151.

Female Guinea Worm.
Natural size.

potash (1 : 3) added, the fungi become clearly visible at the end of ten to fifteen minutes, after the epidermis cells have swollen and become transparent. They form round heaps of spherical conidia, with a diameter of 0.005–0.007 mm. They contain not infrequently a nucleus-like structure or granular protoplasm (vide Fig. 152). Mycelium threads are also present. The older ones contain transverse partitions and one or more nuclei in the individual subdivisions. In some places, the round conidia sprout into mycelium threads; in others, conidia sprout from the latter.

The patches of pityriasis versicolor are almost always found on covered portions of the body. They generally appear first on the chest, then extend gradually to the neck, abdomen, and back. They are sometimes found in the axillæ, on the mons veneris, in the inguinal folds, and on the inner surface of the thighs. In exceptional cases, they are present in the face, never on the hands and feet.

Large portions of the integument are often changed in the manner described, as a result of the coalescence of smaller patches. The centre

Fig. 152.

Microsporon furfur. *a.* Mycelium threads ; *b.* Conidia ; *c.* Epithelium cells.

of the patches sometimes clears up, which the change continues to extend at the periphery.

Subjective symptoms are entirely absent, or there is slight itching, especially while perspiring.

The disease does not occur during childhood or old age. Exacerbations and remissions are noticeable, and sometimes the disease disappears temporarily. Koebner succeeded in inoculating the fungus upon his own integument and upon that of rabbits; but infection from man to man is rarely observed in practice.

II. DIAGNOSIS.—The ready desquamation of the pigment patches distinguishes this from most other pigment changes of the skin, and, in addition, the fungus is readily found with the microscope.

III. TREATMENT.—This consists chiefly of cleanliness. The skin should be rubbed, for several successive evenings, with sapo viridis or with:

R. Sapon. virid.,
  Lact. sulphur.,
  Picis liquid.,
  Spts. vin. dilut...................................... āā ℥ i.
M. D. S. To be rubbed in at night,

and in the morning it should be carefully washed with a woollen cloth. No spot should be overlooked, since it will serve as a fresh starting-point for the disease.

2. *Favus.*

(*Tinea favosa. Porrigo favosa. Dermatomycosis achorina.*)

I. SYMPTOMS AND ANATOMICAL CHANGES.—The fungus of favus (Acharion s. Oidium Schoenleinii) is found almost always on the scalp. When present on other parts, it starts, as on the scalp, from the hair-follicles. It rarely affects the nails (onychomycosis favosa).

The disease appears at first as yellow points (as large as a pin's head) beneath the epidermis; they are perforated by a hair. The spots gradually increase to the size of a pea, bean, or ten-cent piece. At the same time, a central umbilication appears, while the periphery becomes elevated, and the so-called favus cup is thus shaped like a plate.

FIG. 153.

Achorion Schoenleinii from the lower layers of the favus cup. After Kaposi.

When the favus cups are separate, the disease is known as favus dispersus; when they coalesce, as favus confertus. In old cases, the entire scalp may be attacked. The deposits on the skin not infrequently lose their original sulphur color, and become whitish-yellow, light gray, or dirty gray.

If the favus crusts are removed, they may be ground between the fingers, and emit a peculiar mouldy, musty odor. If particles are treated with water or potash, and examined under the microscope, they are found to consist, apart from a few epidermis cells, fat drops, granular detritus, and schizomycetes, of numerous gonidia and mycelium threads of Achorion Schoenleinii (Fig. 153). The gonidia predominate in the lower layers of the crusts; the mycelium threads, in the upper layers. The gonidia are round, band-shaped, or cask-shaped, and are found singly or in groups or rows, while the mycelium threads are segmented, and in places branched.

Achorion Schoenleinii belongs to the mould fungi (hyphomycetes). It can be conveyed to other individuals. The old view that it is identical with the fungus of pityriasis versicolor and herpes has been abandoned.

The increasing development of the favus crusts is attended with important changes in the hairs and epidermis. At first the fungus develops only in the funnel-shaped space situated at the opening of the hair-follicle, so that it is surrounded like a ring by epidermis. But it soon spreads deeper into the follicle, and gives rise to nutritive disturbances

Fig. 154.
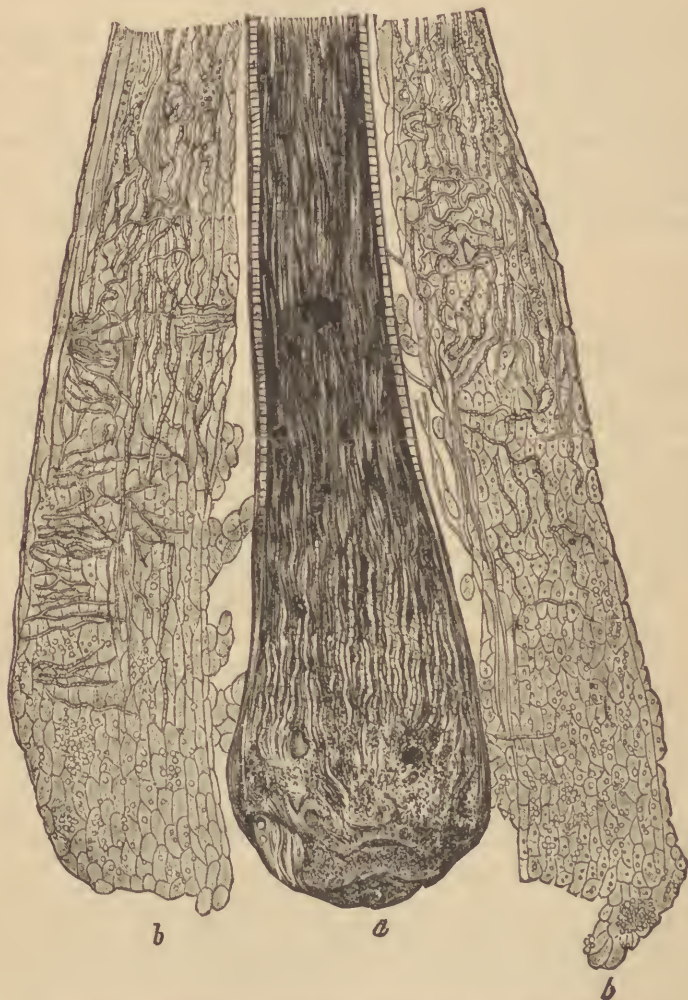
Hair and root sheaths, in favus, infiltrated with conidia and mycelia.

in the hair, partly as the result of pressure, partly by destruction of the hair-papilla. The fungus passes between the external and internal root-sheaths of the hair, later it perforates the upper cuticle of the hair, and enters the cortical layers. The mycelia predominate in this region (vide Fig. 154). The hairs appear dry, destitute of gloss, break readily, can be readily removed on slight traction, or fall out spontaneously. If

the papilla has been destroyed, regeneration of the hair is impossible, and the part remains permanently bald.

If a crust is removed in the earlier stages, the cutis is often found to be red and slightly moist. Later it is covered with young epidermis, so that the removal of the crust shows a delicate, glistening epidermis, which looks almost like a cicatrix. Atrophy of the sebaceous glands, cystoid formations in them, in rare cases cutaneous ulcerations have also been observed.

The development of favus is associated with itching. Eczema of the scalp and swelling of the adjacent lymphatic glands are sometimes observed as complications. In a few cases herpes tonsurans and favus have been noticed in the same individual.

In filthy persons, favus may last thirty years or more, the masses of the fungus accumulating above each other in thick crusts.

If favus develops on other parts of the body, the crusts generally fall off at an earlier period and spontaneous recovery occurs, except in rare cases.

In favus of the nails (onychomycosis favosa), the nails are thickened, fissured, brittle, and destitute of gloss, are exfoliated in parts, and on the addition of potash are found to contain Achorion Schoenleinii. The infection probably is the result of scratching the scalp which is covered with favus.

II. ETIOLOGY.—The disease is either conveyed from animals, or from man to man. It is often found in mice, but the fungus is generally conveyed to man by the cat, the latter being infected by mice or rats. Favus has also been observed in dogs, rabbits, chickens, cows. The greater the degree of uncleanliness the greater is the danger of infection of one individual by another. The disease is most frequent in children or young people, and also in the male sex.

III. DIAGNOSIS.—The diagnosis is rendered extremely easy by the aid of the microscope, so that a mistake is hardly possible.

IV. PROGNOSIS.—The prognosis is good, except that the hair will not be restored in parts which have become bald, if the hair follicles have been destroyed.

V. TREATMENT.—In favus of the scalp, the crusts should be oiled for two hours, and then covered with a piece of flannel dipped in oil, until the crusts can be entirely removed. The scalp is then carefully washed morning and evening with green soap. After each wash, all unhealthy looking hairs should be removed with the fingers or a pair of tweezers, together with all the hairs in the immediate vicinity of the former crusts (in order to prevent reinfection from the fungus which has been left in the follicles). The parts are then rubbed with corrosive sublimate and alcohol (gr. iij. : ℥ iiiss.). This procedure should be continued four to twelve weeks, and the patients should be kept under observation for weeks afterwards, in order that the treatment may again be instituted if a relapse occurs.

Other parasiticides have also been recommended, such as carbolic and salicylic acids, sulphur, tar, creasote, benzin, Peruvian balsam, styrax, turpentine, etc.

In favus of the nails, the affected parts should be excised and painted with corrosive sublimate collodion (1 : 20).

3. *Herpes tonsurans.*

(Tinea tondens. Trichomyces s. Dermatomycosis tonsurans.)

I. ANATOMICAL CHANGES.—The fungus of herpes tonsurans (tricophyton tonsurans) is found not alone on the scalp, but also upon parts which are covered with downy hairs. In the beard, it gives rise to sycosis parasitaria (page 336), and in parts of the skin which are constantly protected and kept moist, it gives rise to eczema marginatum. It also develops occasionally in the substance of the nails (onychomycosis tonsurans).

Upon the scalp and on most other parts of the skin, the changes produced by the fungus appear in three forms, herpes tonsurans vesiculosus, maculosus, and squamosus. The latter is a later stage of the other two.

FIG. 155.

Epidermis scale in herpes tonsurans squamosus, containing more mycelia than conidia. After Kaposi.

Herpes tonsurans vesiculosus is attended at first by the development of small, clear, cloudy, or even purulent vesicles, which are generally not larger than a pin's head. These soon dry into thin scales, at the periphery of which new vesicles appear in a circle, and this may continue until a surface larger than the palm of the hand is affected. While vesicles are still distinct in the peripheral zones, the central portions show the changes of herpes tonsurans squamosus; indeed, the centre itself may have entirely recovered. Adjacent circles may come in contact and form chain-like figures. In old and neglected cases, the larger part of the surface of the body may be affected.

In herpes tonsurans maculosus we notice, at first, small pale-red or brownish-red patches, which are slightly elevated above the healthy skin. While the patch pales and becomes covered with thin scales, a new red zone appears at the periphery, and this process is repeated as in the vesicular form.

The fungus must be looked for in the peripheral zones. In herpes

squamosus, they are contained in the scales; in herpes vesiculosus, in the epidermal covering of the vesicles, but repeated examination is often necessary in order to discover them. In order to make the microscopical preparation transparent, a solution of potash (1 : 3) should be added.

Like the fungus of favus, that of herpes tonsurans appears in the form of round, oval, cask-shaped, homogeneous, granular, or nucleated gonidia and thread-shaped mycelia (Fig. 155), but the latter are more abundant than in the favus fungus. They are often branched, and filled with granular protoplasm, vacuoles, or nuclei. The fungus is situated between the cells of the stratum corneum and the upper cells of the rete Malpighii.

FIG. 156.

Hair in herpes tonsurans. After Kaposi.

Herpes tonsurans of the scalp occurs most frequently in the squamous form. The circles often extend to the integument of the forehead or nape of the neck. Within the circles the hairs break very readily, so that spots which are almost bald are found upon the scalp. The hairs are readily removed by traction, and, under the microscope, it is found that the fungus has penetrated between the root sheaths of the hair follicle, and also into the cortical substance of the hair (vide Fig. 156). As a rule, the papilla is not destroyed, so that the hair is restored, after removal of the fungus.

Onychomycosis tonsurans causes opacity, thickening, fissuring of the nail which may finally be exfoliated. It affects only the finger nails, probably as the result of self-infection in scratching herpetic patches. The herpes on the skin is sometimes recovered, although onychomy-

cosis continues. The fungus elements are distinctly visible, under the microscope, in nail scrapings which have been cleared up by the addition of potash (vide Fig. 157).

Sycosis parasitaria has been discussed on page 336. Huebner has shown that the eczema marginatum of Hebra is nothing more than herpes tonsurans. It is observed most frequently on the inner surface of the thigh and scrotum, and may extend to the pubes and perineum. It occurs more rarely in the axillae, the folds under the breast, umbilicus, or other parts. It forms hyperæmic patches, which are covered with vesicles, pustules, scales, and crusts, and have a zigzag, sharp border. The microscope reveals fungi in the scales. but, according to Hebra, they never penetrate into the hair follicles. The peculiar appearance of eczema marginatum is probably owing to the fact that the diseased

Fig. 157.

Onychomycosis tricophytina. Potash preparation. After Kaposi.

parts, on account of their protected position, are constantly bathed in a warm, moist atmosphere, or that herpes tonsurans complicates a pre-existing eczema.

The disease may last for years, but spontaneous recovery sometimes takes place.

The subjective symptoms consist merely of itching, and even this is not constant.

II. Etiology.—Inoculation proves that the disease is produced by the fungus. Infection from man to man is more frequent than in favus, and the disease occurs endemically in families, schools, and barracks. It is sometimes conveyed from barber shops through the medium of unclean shaving utensils. In other cases, it is contracted from animals (dog, cat, horse, cow, rabbit). The disease has also been observed in individuals who live in damp apartments, constantly use damp underclothing or bedding, or warm poultices. It is observed most frequently

in the spring and autumn, and in damp weather, and chiefly attack children and young people.

III. DIAGNOSIS, PROGNOSIS, TREATMENT.—The diagnosis is rendered positive by the discovery of the fungus. The prognosis is good, the treatment similar to that of favus, but recovery is effected more rapidly.

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